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CLINICAL CHEMISTRY

BARTLEY

BY THE SAME AUTHOR

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MANUAL

OF

CLINICAL CHEMISTRY

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BY

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Thirty-three Illustrations

PHILADELPHIA
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PREFACE.

In this small volume will be found the essentials of chemical diagnosis; or a description of all those chemical processes most useful to the practising physician. It is made up of the last eighty-eight pages of the "Text-book of Medical and Pharmaceutical Chemistry," with the addition of a chapter of Notes on Urinary Diagnosis, and a collection of well-selected experiments with carbohydrates, fats, proteids, and milk, and a scheme for the qualitative analysis of commercial prepared foods.

The Notes on Urinary Diagnosis are essentially the skeleton notes of a course of lectures given for some years, in connection with the course in urinary analysis, at the Long Island College Hospital. The experimental study of the proximate principles of foods, presented at the beginning of the book, is warranted, the author thinks, by the great importance of dietetics in general, and especially of infant feeding. The experimental study of these substances should be undertaken before taking up the more difficult quantitative examination of milk, gastric contents, and urine. It is recommended that these experiments be taken up in connection with the descriptive text, or in connection with the lectures or recitations upon these substances.

As a large part of this book is printed from the same plates used in printing the "Text-book," no apology is needed for the appearance here of some material that does not properly belong in a laboratory manual. The object in binding this part of the "Text-book" separately was to furnish my own students with a small, handy volume, containing the essentials of a suitable course of laboratory work for the second year of medical study, and the physician with a small book on chemical diagnosis, as applied to the gastric contents, milk, feces, and urine. The author would here acknowledge his indebtedness to Haliburton's "Essentials of Chemical Physiology" for many suggestions in preparing the course of experimental study of the proximate principles of foods.

E. H. B.

November, 1898.

THE QUALITATIVE DETECTION OF THE COMMON SUGARS. (*Allen.*)

	DEXTROSE, $C_6H_{12}O_6$.	LEVULOSE, $C_6H_{12}O_6$.	MILK-SUGAR, $C_{12}H_{22}O_{11} + H_2O$.	MALTOSE. $C_{12}H_{22}O_{11} + H_2O$.	CANE-SUGAR, $C_{12}H_{22}O_{11}$.	DEXTRIN. $(C_6H_{10}O_5)_n$.
1. Moisten the solid sugar with water, and stir in the cold with concentrated sulphuric acid (1.845 sp. gr.).	Not affected when pure.	Not affected when pure.	Not affected.	Slightly reddish or brownish, gradually turning darker.	Charred.	Not affected.
2. Triturate the solid sugar with caustic soda, or boil it with a 3 per cent. caustic soda solution for one minute.	Deep-brown coloration.	Deep-brown coloration.	Not affected.	Slightly discolored.	Not affected.	Not affected.
3. To the neutral aqueous solution add a few drops of Fehling's copper solution, and heat to boiling for a few minutes.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Not affected.	Not affected.
4. Heat the solution to boiling for half an hour with one-twentieth of its bulk of strong sulphuric acid, neutralize with soda, and heat to boiling with Fehling's solution for a few minutes.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	Red precipitate of Cu_2O
5. To a few drops of Fehling's solution add caustic soda and ammonia, heat to boiling, and add the saccharine solution drop by drop, keeping the liquid boiling (Pavy's test).	Liquid decolorized.	Liquid decolorized.	Liquid decolorized.	Liquid decolorized.	No change.	No change.
6. Boil the solution for two minutes with 1 c.c. of a liquid containing 4 per cent. of cupric acetate, and 1 per cent. of acetic acid ($C_2H_3O_2H$).	Red precipitate of Cu_2O.	Red precipitate of Cu_2O.	No change.	No change.	No change.	No change.
7. Observe the solution in the polarimeter.	Dextro-rotatory.	Levo-rotatory.	Dextro-rotatory.	Dextro-rotatory.	Dextro-rotatory.	Dextro-rotatory.
8. Heat the solution with diluted acid, as in test 4, and observe again in the polarimeter.	Dextro-rotatory power unchanged.	Levo-rotatory power unchanged.	Dextro-rotatory power increased.	Dextro-rotatory power diminished.	Dextro-rotatory power changed to levo-rotatory.	Dextro-rotatory power diminished.

CLINICAL CHEMISTRY.

EXERCISES IN EXPERIMENTAL PHYSIOLOGICAL CHEMISTRY.

The student should refer to the descriptive part of the text-book for the subjects treated of here.

THE CARBOHYDRATES.

1. Note the general appearance of the specimens of grape-sugar or dextrose, dextrin, and starch which are passed around.

2. Put some of each into cold water. Starch is insoluble; dextrose and dextrin slowly dissolve, but more readily in hot water.

3. *Apply Trommer's Test for Dextrose.*—Put a few drops of copper sulphate solution into a test-tube, then solution of dextrose, and then strong sodium hydroxide. On adding the NaOH a precipitate is first formed, which, on adding more, redissolves, forming a blue solution. On boiling this, a yellow or red precipitate (cuprous hydrate or oxide) forms.

4. *Dextrin.*—Add iodine solution to the solution of dextrin, and a reddish-brown color is produced. The color disappears on heating.

5. *Starch.*—(*a*) Examine microscopically the scrapings from the surface of a freshly cut potato. Note the appearance of the starch-grains, with their concentric markings. (See table, p. 369.)

   (*b*) On boiling starch with water, an opalescent solution is formed, which, if strong, gelatinizes on cooling.

   (*c*) Add iodine solution. An intense blue color is produced, which disappears on heating, and, if not heated too long, reappears on cooling. N. B.—Prolonged heating drives off the iodine, and consequently no blue color returns on cooling.

   (*d*) Conversion into dextrin and dextrose. To some starch solution in a flask add a few drops of 25 per cent. sulphuric acid and boil for fifteen minutes. Take out a portion of the liquid at intervals of five minutes and test for dextrin and dextrose.

6. *Glycogen.*—Solution of glycogen is given round: (*a*) It is opalescent, like the solution of starch.

(*b*) With iodine it gives a brown color like dextrin.

(*c*) By boiling with 25 per cent. sulphuric acid it is converted into dextrose.

ACTION OF MALT UPON STARCH; MALTOSE.

1. Prepare a 0.5 per cent. solution of starch.

2. Prepare some malt extract by digesting 10 gm. of powdered malt with 50 c.c. of water at 50° C. for three hours, and filter through absorbent cotton. This extract contains the diastasic or malting ferment.

3. To the starch solution add one-tenth of its volume of malt extract, and place the mixture in a water-bath at 40° C. From time to time test portions of the liquid by mixing a drop of it with a drop of iodine solution on a testing slab. The blue color at first seen is soon replaced by a violet (mixture of blue and red), and then by a reddish-brown (due to *erythrodextrin*), which gradually vanishes. Alcohol added to the liquid when all starch and erythrodextrin have gone, still causes a precipitate of a dextrin, which, as it gives no color with iodine, is called *achroodextrin*. The liquid also contains a reducing sugar—maltose.

4. Treat the solution of starch, as before, with one-tenth of its volume of malt extract, and keep it in the warm chamber at 40° C. for three hours.

(*a*) Take 50 c.c. of the product (which is a solution of maltose) and determine how much of it is necessary to reduce 10 c.c. of Fehling's solution. (See p. 653.)

(*b*) Take another 50 c.c. and boil it in a flask with 5 c.c. of hydrochloric acid for half an hour. This converts maltose into dextrose. After cooling, bring the liquid to its original volume (50 c.c.) by adding water, and again determine its increased reducing power with Fehling's solution. If $x =$ c.c. of maltose solution necessary to reduce 10 c.c. of Fehling's solution, then $\frac{2x}{3} =$ c.c. of dextrose solution necessary for the same purpose. Or, the ratio of the reducing power of maltose to dextrose should be as 66 to 100. The strength of the maltose solution can be calculated from the fact that 10 c.c. of Fehling's solution corresponds to 0.05 gm. of dextrose.

FATS.

Tests to be practised upon pure lard.

1. Note that it is insoluble in water. Test its solubility in ether, chloroform, and petroleum-ether.

2. Boil a small portion with NaOH solution. It dissolves and yields a solution of soap.

3. Add to this solution a few drops of 25 per cent. sulphuric acid. On heating, a layer of fatty acid collects on the surface.

4. Filter the acidulated solution obtained in 3; evaporate a small portion of the filtrate to dryness. Heat cautiously to about 160° C. with two drops of carbolic acid, dissolve the residue in water, and add ammonia. A carmine color shows the presence of glycerin.

THE PROTEIDS.

(See p. 503.)

Tests for Proteids.—The following tests are to be tried with a mixture of one part of white of egg to ten of water. (Egg-white contains a mixture of albumin and globulin.)

1. *Heat Coagulation.*—Faintly acidulate with a few drops of 2 per cent. acetic acid and boil. The proteid is rendered insoluble (coagulated proteid).

2. *Precipitation with Nitric Acid.*—The addition of strong nitric acid to the original solution also produces a white precipitate.

3. *Xanthoproteic Reaction.*—On boiling, the white precipitate produced by nitric acid turns yellow; after cooling, add ammonia; the yellow color changes to orange.

4. *Millon's Test.*—Millon's reagent, which is a solution of the nitrate of mercury containing excess of nitric acid (see p. 503), gives a white precipitate, which turns brick-red on boiling.

5. *Bromine water* precipitates all forms of proteids from solutions acidified with HCl.

6. After the addition of acetic acid, potassium ferrocyanide gives a white precipitate.

7. Add a drop of a 1 per cent. solution of cupric sulphate to the original solution and then caustic potash; a violet solution is obtained.

8. Repeat experiment 6 with a solution of commercial peptone, and note that a rose-red (biuret) color is obtained.

9. **Action of Neutral Salts.**—The following experiments may be performed with serum, which, like egg-white, contains albumin and globulin:

(*a*) Saturate it with magnesium sulphate, by adding crystals of the salt to the serum and shaking vigorously in a flask. A white precipitate of serum-globulin is produced. Filter. The filtrate contains serum-albumin. (Serum-globulin is incompletely precipitated by diluting the serum with a large quantity of water, and also by adding weak acetic acid to, or passing a stream of carbonic acid gas through, diluted serum.)

(*b*) Saturate another portion with ammonium sulphate or zinc sulphate; a precipitate is produced of both the globulin and albumin. Filter. The filtrate contains no proteid.

10. Repeat the last experiment with a solution of commercial peptone. A precipitate is produced of the albumoses or proteoses it contains. Filter. The filtrate contains the true peptone. Ammonium sulphate and zinc sulphate precipitate all proteids except peptone.

THE ALBUMINATES AND ALBUMINOIDS.

(See pp. 510 and 519.)

1. *Action of Acids and Alkalies on Albumin.*—Take three test-tubes and label them A, B, and C.

In each place an equal amount of diluted egg-white, similar to that used above.

To A add a few drops of 0.1 per cent. solution of NaOH.

To B add the same amount of 0.1 per cent. solution of NaOH.

To C add a rather larger amount of 0.1 per cent. sulphuric acid.

Put all three into the warm bath at about the temperature of the body (36°–40° C.).

After five minutes remove test-tube A and boil. The proteid is no longer coagulated by heat, having been converted into *alkali-albumin.* After cooling, color with phenol-phthaleïn solution and neutralize with 0.1 per cent. acid. At the neutral point a precipitate is formed which is soluble in excess of either acid or alkali.

Next, remove B. This also now contains alkali-albumin. Add to it a few drops of sodium phosphate, color with phenol phthaleïn, and neutralize as before. Note that the alkali-albumin now requires more acid for its precipitation than in A, the acid first added converting the sodium phosphate into acid sodium phosphate.

Now remove C from the bath. Boil it. Again there is no coagulation, the proteid having been converted into *acid-albumin.* After cooling, color with phenol-phthaleïn and neutralize with 0.1 per cent. NaOH. At the neutral point a precipitate is formed, soluble in excess of acid or alkali. (Acid-albumin is formed more slowly than alkali-albumin, so it is best to leave this experiment until the last.)

2. Dissolve some gelatin in hot water. On cooling, the solution sets into a jelly (gelatinization).

3. Saturate separate portions of a dilute solution of gelatin with (*a*) ammonium sulphate, (*b*) sodium sulphate, (*c*) magnesium sulphate, (*d*) sodium chloride.

4. Add a few drops of acetic acid to some saliva. A stringy precipitate of mucin is formed.

5. To another portion add alcohol. A precipitate forms. Try its solubility in lime-water.

Albumoses and Peptones.

Use a solution of somatose, or of Mosquera beef-meal.

Make a solution of one of these substances in 10 per cent. sodium chloride solution, and filter. Very little residue may be left on the filter. This may consist of dysalbumose, an insoluble form of hetero-albumose, formed during the process of preparing the substance, or of unchanged proteid. If hot saline solution is used instead of cold, as a solvent, this amount of insoluble residue is increased, hetero-albumose being to a slight extent precipitated by heat.

The solution gives the following tests:

(*a*) Biuret reaction (due both to peptone and albumoses). (See 6 and 7, under Proteids.)

(*b*) A drop of nitric acid gives a precipitate, which dissolves upon warming and reappears on cooling. (This is due to the primary albumoses present.)

(*c*) It does not coagulate on heating.

Otherwise it gives the general proteid reactions. (See p. 503.)

6. For the separation of the albumoses and peptone proceed as follows:

(*a*) Saturate the solution with ammonium sulphate or zinc sulphate and filter. The filtrate contains the peptone and the precipitate the albumoses. The peptone is not precipitated by nitric acid, nor by most of the reagents that precipitate other proteids. It is precipitated completely by alcohol, tannin, and potassio-mercuric iodide; imperfectly by phospho-tungstic and phospho-molybdic acids. It gives the biuret reaction, but in the presence of ammonium sulphate a large excess of caustic potash is necessary.

(*b*) Dialyse another portion of the solution; hetero-albumose is precipitated.

(*c*) Saturate another portion of the solution with sodium chloride after faintly acidulating with acetic acid. Proto-albumose and hetero-albumose are precipitated. Filter. The filtrate contains the deutero-albumose and peptone.

The proto- and hetero-albumose may be redissolved by adding distilled water, and may be separated from each other by dialysis (see *b*).

Deutero-albumose may be separated from the peptone by saturating the solution with ammonium sulphate or zinc sulphate, or by the addition of a crystal of glacial phosphoric acid. These reagents precipitate the deutero-albumose, but not the peptone.

Deutero-albumose gives the nitric-acid reaction (see 5, *b*) characteristic of the albumoses only in the presence of excess of salt. If the salt is removed by dialysis, nitric acid then causes no precipitate.

MILK.

A. **Milk.**—1. Examine a drop of milk with the microscope. Allow some milk to stand for three hours, and siphon off the lower two-thirds with a rubber tube. Use the skim-milk thus drawn off for the tests.

2. Note the specific gravity of fresh milk with the lactometer, and observe that the specific gravity is increased by the removal of the lightest constituent—the cream. Compare the specific gravity of the top rich milk with the skim-milk drawn off.

3. The reaction of fresh milk is usually neutral or slightly alkaline.

4. Warm some milk in a test-tube, to the temperature of the body, and add a few drops of *liquid rennet* or *essence of pepsin*. After standing, a *curd* is formed from the conversion of *caseinogen*, the chief proteid in milk, into *casein*. The casein entangles the fat globules; the liquid is termed *whey*. No curd forms if the rennet solution is previously boiled. Heat kills ferments.

5. To another portion of warm milk diluted with water, add a few drops of strong acetic acid. A lumpy precipitate of caseinogen entangling the fat is formed.

6. Filter off the curd, and test the whey for *lactose* or *milk-sugar* by Trommer's test; for *lactalbumin* by boiling, or by Millon's reagent; and for earthy (that is, calcium and magnesium) *phosphates* by ammonia, which precipitate these phosphates.

7. *Fat* (*butter*) may be extracted from the curd by shaking it with ether or petroleum-ether; on evaporation of the ethereal extract the fat is left behind, orming a greasy stain on paper.

8. *Caseinogen*, like the globulins, is precipitated by saturating milk with sodium chloride or magnesium sulphate, but differs from the globulins in not being coagulated by heat. The precipitate produced by saturation with salt floats to the surface with the entangled fat. The clear, salted whey is seen below after an hour or two.

COAGULATION OF MILK.

9. Prepare a solution of pure caseinogen as follows: Saturate milk with magnesium sulphate, by shaking it with excess of the powdered salt. Allow it to stand for a few hours and then filter. The caseinogen and fat remain together on the filter. Save the filtrate and label it A. Wash the precipitate on the filter with a saturated solution of magnesium sulphate, until the washings contain no albumin. Add water to the precipitate. The caseinogen dissolves, the fat being insoluble. In this way a solution of caseinogen in weak magnesium sulphate is obtained. So far the operations should be performed beforehand by the demonstrator.

10. To this solution add acetic acid. The caseinogen is precipitated; collect it on a filter; wash the acid away with distilled water. Dissolve the precipitate in lime-water by grinding it up in a mortar with the lime-water; filter, and an opalescent solution of caseinogen is obtained.

11. To a portion of this solution add a few drops of rennet extract. Put it in the water-bath at 40° C., and, if the caseinogen has been thoroughly washed, no coagulation will occur.

12. Treat another portion in the same way, adding, however, a few drops of 0.5 per cent. phosphoric acid as well as the rennet. Warm to 40° C. Coagulation—that is, formation of casein from caseinogen—usually occurs in a few minutes.

13. Examine the filtrate A (see 9). Saturate a portion with sodium chloride. A small amount of precipitate of a proteid comes down. This is the so-called lacto-globulin. Milk contains only a trace of true globulin. The precipitate is mostly caseinogen previously left in solution, together with calcium sulphate.

14. Heat another portion of A to 77°, acidifying faintly with a few drops of 2 per cent. acetic acid. Lactalbumin is coagulated at this temperature.

15. Ringer's method of showing the conversion of caseinogen into casein: Milk is strongly acidified with acetic acid. This precipitates the caseinogen and entangled fat. The precipitate is collected on a filter, thoroughly washed with distilled water, and ground up in a mortar with calcium carbonate. The mixture is thrown into excess of distilled water. The fat rises to the top; the excess of calcium carbonate falls to the bottom. The intermediate fluid contains the caseinogen in solution; it is usually very opalescent. Take some of this solution and divide it into three parts—A, B, and C.

To A add rennet.

To B add a few drops of a 10 per cent. solution of calcium chloride.

To C add both rennet and calcium chloride.

Put all three in the water-bath at 40° C. A clot of casein forms in C, but not in A and B.

16. Prepare some whey as under 4, and heat it to 70° C. Mix together two volumes of the whey and one volume of the rich top milk. Dissolve in this mixture milk-sugar in the proportion of 0.5 gm. to 25 c.c. (See p. 617.) Compare this mixture with whole milk, and with milk diluted with twice its volume of water by tests 4 and 5, above.

17. Filter some milk through a clay filter * by means of a Bunsen's filter-pump. Fit a doubly perforated rubber stopper into the open end of a porous battery-cup. Fit two glass tubes into the stopper, one closed by a pinch-cock and the other connected with the pump. Set the cup into some milk in a beaker and apply suction. After a time the cup will contain a clear milk-serum, or whey, containing lactose, lactalbumin, and salts. Caseinogen and fat do not pass through. Test the clear fluid for proteids, lactose, and salts.

SALIVA.

Collect some saliva by chewing a small quantity of paraffin and expectorating in a suitable vessel.

1. To a little saliva in a test-tube add acetic acid. Mucin is precipitated in stringy flakes.

2. Filter some fresh saliva, to separate cells and mucus, and apply the xanthoproteic or Millon's test to the filtrate; the presence of proteid is shown.

3. Put some 0.5 per cent. starch solution into four test-tubes. Add some filtered saliva to A. To B add saliva and three drops of HCl; to C add 1 c.c. of NaOH solution; to D add saliva, and boil. Put all the tubes in the water-bath at 40° C. After ten minutes, remove and test them with iodine, and Trommer's test. The saliva in A will be found to have converted the starch into dextrin and maltose. In B and C no sugar is formed, because strong acids and alkalies arrest the action of saliva on starch; nor in D, because the diastase was destroyed by heat.

4. The presence of potassium sulphocyanide (KCNS) in saliva may be shown by the red color given by a drop of ferric chloride. This color is discharged by mercuric chloride.

5. The reaction of saliva is usually alkaline to litmus paper.

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* The Chamberlain or Berkfeld filter answers very well, or we may use the porous cup used in the Bunsen cell.

PEPTIC DIGESTION.

1. Half fill four test-tubes—
A with water.
B with 0.2 per cent. hydrochloric acid.
C with 0.2 per cent. hydrochloric acid.
D with solution of white of egg (1 to 9 of water).

2. To A add 1 c.c. of a two per cent. solution of a good pepsin, and a piece of a solid proteid like fibrin or coagulated egg-white.

To B also add 1 c.c. of the pepsin solution and a piece of fibrin or coagulated egg-white.

To C add only a piece of fibrin.

To D add 1 c.c. of pepsin solution and fill up the tube with 0.2 per cent. hydrochloric acid.

3. Put all four tubes into the water-bath at 40° C., and observe them from time to time.

In A the albumin (or fibrin) remains unaltered.

In B it becomes swollen, and gradually dissolves.

In C it becomes swollen, but does not dissolve.

4. Examine the solution in test-tube B.

(*a*) Color some of the liquid with phenol-phthaleïn and neutralize with dilute alkali. Acid-albumin, syntonin, or parapeptone is precipitated.

(*b*) Take another test-tube and put into it a drop of 1 per cent. solution of copper sulphate; empty it out so that the merest trace of copper sulphate remains adherent to the wall of the tube; then add the solution from test-tube B and a few drops of strong caustic soda. A pink color (biuret reaction) is produced. This should be carefully compared with the violet tint given by unaltered albumin.

(*c*) To a third portion of the fluid, in test-tube B, add a drop of nitric acid; albumoses or propeptones are precipitated. This precipitate dissolves on heating and reappears on cooling.

5. These three tests should be repeated with the digested white of egg in test-tube D.

PANCREATIC DIGESTION.

1. A 1 per cent. solution of sodium carbonate, to which a little glycerine extract of pancreas has been added, forms a good artificial pancreatic fluid. In place of this we may use one of the commercial solutions of pancreatic extract, diluted with an equal volume of 1 per cent. Na_2CO_3.

2. Half fill three test-tubes with one of these solutions:

A. To this add half its bulk of diluted egg-white (1 in 10).
B. To this add a piece of fibrin.
C. Boil this, cool, then add fibrin.

3. Put all into the water-bath at 40° C. After half an hour, test A and B for alkali-albumin by neutralization with dilute acetic acid, and filter. Boil the filtrate to precipitate any unchanged albumin, and filter again. Test the filtrate for albumoses by 6, p. 13, and for albumoses and peptone by the biuret reaction.

4. Note that the fibrin in B does not swell up and dissolve as in gastric digestion, but that it is eaten away from the edges to the interior.

5. In C no digestion occurs, as the ferments have been destroyed by boiling.

6. Add equal quantities of a solution of starch to three test-tubes:
D. To this add a few drops of pancreatic extract.
E. To this add a few drops of bile.
F. To this add both bile and pancreatic extract.

7. Put these into the water-bath and test small portions of each every half minute by the iodine reaction. The blue color-reaction disappears first in F, then in D, while E undergoes no change. Now test D and F for maltose by Trommer's test.

8. Shake up a few drops of olive oil with the above artificial pancreatic juice. A milky fluid (emulsion) is formed, from which the oil does not readily separate on standing. Repeat this experiment with the addition of one-tenth volume of bile.

9. The foregoing experiments illustrate the action of pancreatic juice on all three classes of foods.

THE EXAMINATION OF ARTIFICIAL FOODS.

The following scheme will serve as a general guide in the examination of prepared foods for the detection of the proximate principles. For confirmatory tests the student should consult the preceding exercises:

1. If the food be a liquid, dilute and apply the tests at once. If a powder or paste, proceed as follows: Shake a portion of the food in a test-tube with ether, evaporate the ether, and examine the residue for fat or oil globules. If fat is found to be present, shake a portion of the food repeatedly with ether, pouring off the ether each time before adding fresh ether, until the fat is all removed. Warm, to drive off the ether, and shake about 5 gm., if a powder or paste, with 50 c.c. of cold water. Label **cold solution.**

Note the color, odor, transparency or opalescence, taste, smell, solubility, reaction, etc. *This solution can not contain uncooked starch,* *fibrin, gelatin, or fats. If neutral in reaction, albuminates are absent, and need not be tested for.*

Dark-colored liquids suggest blood, meat extract, malt, etc.

Light-colored liquids suggest milk solids, certain proteids, or carbohydrates.

Opalescence suggests cooked starch in solution, glycogen, or globulins.

2. Filter from the insoluble portion, if necessary. Add iodine to a small portion of both the filtrate and the insoluble residue.

Blue color indicates starch. Confirm by conversion into sugar with saliva or dilute H_2SO_4. (See 5, p. 13.)

Reddish-brown color indicates dextrin or glycogen. Glycogen forms opalescent solutions, precipitated by alcohol and basic lead acetate; dextrin forms a clear solution, is not precipitated by basic lead acetate nor by alcohol, unless in large excess. Both form reducing sugars when boiled with dilute H_2SO_4.

3. Boil a portion of the residue insoluble in cold water, with water; filter while hot and examine the filtrate, when cold. *This solution will contain any starch or gelatin present in the food. It will not contain albumin or globulin, and albuminates only in acid or alkaline solutions.* Test solubility of any insoluble residue with acids and alkalies. Examine with microscope. Examine the cold and hot solutions (prepared by 1 and 3) separately.

4. Add copper sulphate and caustic soda to the aqueous solution.

(*a*) Blue solution: Boil; yellow or red precipitate. **Dextrose, maltose,** or **lactose.** (For distinguishing tests see under Carbohydrates.) (See also p. 369.)

(*b*) Blue solution: Very slight reduction on boiling; boil some of the original solution with dilute sulphuric acid, and then boil with copper sulphate and caustic potash; abundant yellow or red precipitate: **Cane-sugar.**

(*c*) Violet solution: **Proteids** (albumins, globulins, albuminates) and **gelatin.** In presence of magnesium sulphate the NaOH causes also a white precipitate of magnesium hydroxide.

(*d*) Pink solution: biuret reaction. **Peptones** or **albumoses (proteoses).** In presence of ammonium sulphate very large excess of NaOH is necessary for this test. Only a trace of copper sulphate must be used.

5. When proteids are present, proceed as follows: Test the reaction with litmus paper, and neutralize:

(*a*) Neutralization causes a precipitate soluble in excess of weak acid or alkali. **Acid-albumin** or **alkali-albumin,** according as the reaction of the original liquid is acid or alkaline respectively. If the original liquid is neutral, acid-albumin and alkali-albumin must both be absent. A precipitate produced by acid, insoluble in excess, probably caseinogen (see 6).

6. Acidify the filtrate obtained in 5 with a few drops of acetic acid. Caseinogen precipitates, if present. Filter, and boil the filtrate.

(*a*) Precipitate produced: **Albumins** or **globulins.**

(*b*) No precipitate: **Gelatin, proteoses,** or **peptones** may be present.

7. If albumin or globulin be present, remove according to 5.

(*a*) Saturate the filtrate with sodium sulphate or magnesium sulphate. Precipitate: **Gelatin** and **primary albumoses,** if present.

(*b*) Or, better, add one-fourth volume of solution of potassium dichromate. Precipitate: **Gelatin.**

8. Saturate the solution from 7 (*a*), from which albuminates, albumin, globulin, and gelatin have been removed, if present, with ammonium sulphate, or zinc sulphate.

(*a*) Precipitate: **Proteoses.** (*b*) No precipitate: **Peptone** and **meat bases** may be present. If there is a precipitate, filter clear.

If both are present, the precipitate contains the proteoses, and the filtrate the peptone.

9. To a fresh portion of the solution from 7 (*a*), add nitric acid.

(*a*) No precipitate, even though excess of sodium chloride be also added: No **proteoses.**

(*b*) No precipitate, until excess of sodium chloride is added: **Deuteroproteose.**

(*c*) Precipitate which disappears on heating and reappears on cooling: **Proteoses.** This is the distinctive test of all the proteoses or albumoses, and is given by all of them. For one of them, however (deuteroproteose), excess of sodium chloride must also be added.

In all cases nitric acid *plus* heat causes a yellow color, turned orange by ammonia.

10. Remove all proteids from the solution by the addition of HCl and bromine water, or by slightly acidifying with acetic acid and adding a solution of tannin. Filter.

Test filtrate for meat bases and amids:

(*a*) Add excess of ammonia and then silver nitrate. A precipitate: Xanthin bases, creatinin, uric acid, etc.

(*b*) Acidify strongly with HCl and add phosphotungstic acid. The meat bases and amido-acids are precipitated. The precipitate dissolves on heating to 100° C., and reprecipitates on cooling.

THE CLINICAL EXAMINATION OF THE GASTRIC CONTENTS.

Before beginning the study of the methods for the clinical examination of gastric juice, it will be well to consider a few of the principles of volumetric analysis.

Because of the rapidity and ease with which quantitative analyses can be made by means of volumetric solutions, this method is almost universally adopted in clinical work. As most of the quantitative processes described in the following pages of this book depend upon the use of volumetric solutions, it is necessary for the student to understand their preparation, the principles of their use, and the apparatus employed.

Volumetric Quantitative Analysis, or Estimation.—By **volumetric analysis** is meant the quantitative estimation of a substance, by adding to it a measured volume of liquid containing a known amount of the reagent, and depending upon an indicator to show when the precipitation or reaction is completed. This process is called **titration.**

Volumetric analysis requires :

1. A graduated vessel, from which accurately measured portions of the reagent liquid may be delivered, called a **burette.**
2. A solution of the reagent, of known chemical power, and called a **standard** or **volumetric solution.**
3. The decomposition produced by chemical in the standard solution with the substance to be estimated must be either such that by itself or by the aid of an indicator *its completion is unmistakably evident to the eye,* and thus the quantity of the substance with which it has reacted may be calculated.

Measuring Instruments.—The apparatus needed for the volumetric methods are usually few and inexpensive. An expensive balance is not essential for the methods to be mentioned below, as the standard solutions are to be had of wholesale druggists or chemical dealers, or can be prepared by any competent pharmacist. A **burette** is a graduated tube holding from 25 c.c. to 100 c.c., and provided with a stop-cock at one end and terminating in a tube of small caliber. The most convenient burette is one of 50 c.c. capacity, and graduated to 0.1 c.c. (See Fig. 1.)

Pipettes (Fig. 3) are graduated tubes, drawn down at one end to a small opening, and intended to be filled by suction at the upper end, after which the finger is pressed upon the upper end to control the flow. A graduated flask is a glass flask having a narrow neck, upon which is a mark denoting its capacity when filled to this mark. The most convenient sizes for general purposes are 50 c.c., 100 c.c., 250 c.c., and 1000 c.c. It is convenient to have a 10-c.c. and a 25-c.c. graduated cylinder.

Standard Solutions.—A standard solution is a solution of a chemical substance, containing a known amount of the substance in a measured volume of liquid. It is usual to express the strength of such solutions in the number of grams of the active ingredient to the liter, or 1000 c.c., of the solution.

A **normal standard solution** is one made to contain the chemical equivalent of one atom of hydrogen, or any other monad element, expressed in grams, dissolved in one liter.

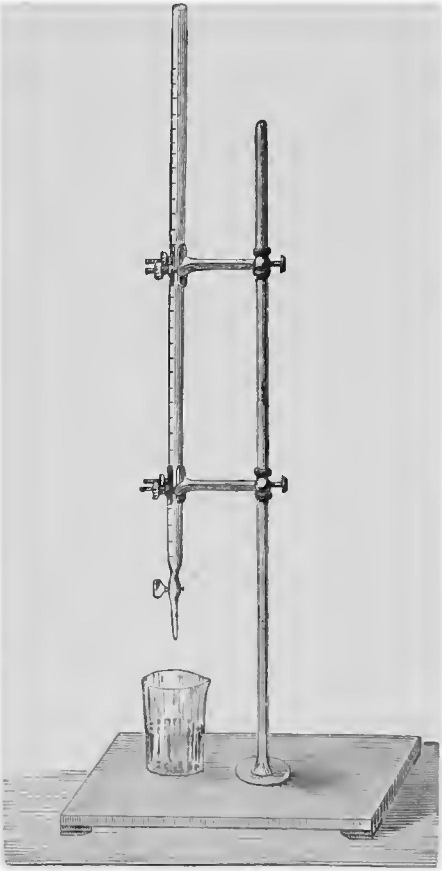

FIG. 1.—GRADUATED BURETTE.

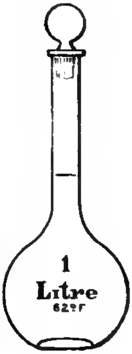

FIG. 2.

FIG. 3.

Thus: HCl is the chemical equivalent of Na, Ag, Br, I, or H

$HCl + AgNO_3 = AgCl + HNO_3$.

$HCl + NaOH = NaCl + H_2O$.

$HCl + NaI = NaCl + HI$.

The molecular weight of HCl is $35.5 + 1 = 36.5$. The normal solution of HCl will then be made to contain 36.5 gm. by weight of HCl to the liter. The chemical equivalent of H_2SO_4 in Na or H is twice that of HCl, or one molecule of H_2SO_4 will neutralize Na_2. Hence the normal solution will contain one-half its molecule, expressed in grams, dissolved in one liter, or $98 \div 2 = 49$ gm. A normal solution of oxalic acid, $H_2C_2O_4 . 2H_2O$, will also contain one-half its molecular weight, dissolved in a liter. A normal solution of sodium hydroxide will contain the molecular weight expressed in grams, because NaOH is the equivalent of HCl, Br, I, or any other monad.

A decinormal solution is one containing one-tenth the active ingredient of the normal solution, or one-tenth the chemical equivalent of hydrogen, expressed in grams, dissolved in a liter. It is often made from the normal solution by diluting 100 c.c. of the latter to a liter with distilled water. A $\frac{1}{50}$ normal and a $\frac{1}{100}$ normal solution or a centinormal solution are sometimes used. A normal solution is frequently expressed thus: $\frac{N}{1}$; a decinormal, $\frac{N}{10}$, or $\frac{1}{10}$N; and a centinormal, $\frac{1}{100}$N, or $\frac{N}{100}$.

Preparation of Decinormal Sodium Hydroxide.—As commercial sodium hydroxide is not of definite composition, it is usually prepared by titration against a normal or decinormal solution of oxalic acid. To prepare a normal solution, weigh out exactly 63 gm. of pure crystallized oxalic acid, $H_2C_2O_4 . 2H_2O$, taking care that it is free from any evidence of extra moisture or efflorescence of the crystals, and dissolve this in one liter of distilled water. Weigh out about 50 gm. of best commercial NaOH, and dissolve this in water, cool, pour into the 1-liter flask and make up to one liter.

To "*standardize*" this solution proceed as follows: Measure out into a beaker 10 c.c. of the solution, add three drops of a 1 per cent. alcoholic solution of phenolphthalein, and run in the oxalic acid from a burette until the pink color is just discharged. Suppose 12 c.c. of the acid solution be required to do this. Then 10 c.c. of the NaOH solution contain the amount of NaOH that should be contained in 12 c.c.; 100 c.c. will contain the amount that 120 c.c. should contain. To make a normal solution of this solution, 20 c.c. of water must be added to every 100 c.c. We may calculate the amount of water to be added to any number of cubic centimeters by the following proportion:

$$10 : 12 : : \text{any measured volume} : \varphi.$$

Or, we may calculate the number of cubic centimeters to be used to make 1000 c.c. by the proportion:

$$10 : 12 : : \varphi : 1000.$$
$$10 \times 1000 = 10{,}000. \quad 10{,}000 \div 12 = 833.3 \text{ c.c.}$$

From this normal NaOH solution we may make a decinormal solution by measuring out 100 c.c., putting this into a liter flask and filling to the mark on the neck with distilled water.

Statement of results of a titration are expressed by two methods: Either by calculating the total amount or the percentage of acids present, or by calculating the number of cubic centimeters of decinormal alkali required to neutralize the acid of 100 c.c. of the fluid. In the case of total acidity, or total organic acids, or in the estimation of acid salts of gastric juice, it is impossible to state the percentage of these causes of acidity, except in equivalents of some acid, as HCl, which is usually selected. Suppose, for example, that the amount of $\frac{N}{10}$NaOH required to give a decided pink color in 10 c.c. of the filtered fluid containing phenolphthalein be 6 c.c.: It would require $6 \times 10 = 60$ c.c. for 100 c.c. of fluid. Each cubic centimeter of the NaOH solution neutralizes 0.00365 gm. of HCl. Sixty c.c. would neutralize an acidity the equivalent of $60 \times 0.00365 = 0.2190$ gm. of HCl in 100 c.c., or 0.22 per cent. Owing to the indefinite character of the above statement, many prefer the second method of stating the results. In the above example, which represents about an average normal digestion, the acidity would be said to be 60. Some would call it 60 degrees, while others would say 60 per cent., meaning 60 per cent. of decinormal alkali solution required for neutralization. The second method is quite generally used, and is in most cases preferable.

Chemical Examination of the Gastric Juice.—The chemical examination of the contents of the stomach has, in recent times, become an important aid in the diagnosis of diseases of the stomach. The clinical examination of the stomach and its contents, for diagnostic purposes, generally includes an estimation of the rapidity of absorption, the motility, the reaction, and acidity of the gastric juice; the determination of the kind and amount of acids present; an examination for syntonin, albumose, and peptone; the determination of the digestive activity, and a microscopical examination of the stomach contents.

The stomach contents for examination are obtained as follows: The usual method is to administer to the patient, on on empty stomach, an ordinary dry roll and a definite quantity—say, 300 c.c., or about ⅔ of a pint—of fluid, either simple warm water or weak tea without milk or sugar. The roll should be of a tolerably uniform weight of about 35 gm., or 540 grs. Such rolls contain about 7 per cent. of proteids, 0.5 per cent. of fat, 0.4 per cent. of sugar, 52.5 per cent. of non-nitrogenous matter, and about 1 per cent. of ash.

The test-meal may consist of oatmeal or barley, cooked in water and flavored with a little salt, and a glass of water. About one hour after taking the test-breakfast the stomach-tube is inserted, and that which remains in the stomach is drawn off. The amount obtained should be about 40 c.c., or $1\frac{1}{3}$ ozs. A greater variation than 15 to 20 c.c. in either direction should be regarded as pathological.

Method of Drawing Out the Contents of the Stomach for Chemical and Microscopical Examinations.—The contents of the stomach are usually drawn off about one hour after the person has taken one of the above test-breakfasts. This is accomplished by the stomach-tube, Einhorn's gastric bucket, or by causing the patient to vomit. The first of these methods is usually to be preferred. Place the patient in a sitting position, dip the rounded end of the elastic stomach-tube in warm water, hold it about six inches from its extremity, and, as the patient opens his mouth, pass it to the back of his throat, and ask him to swallow. As he does so, pass the tube gently but rapidly into the esophagus, and onward to the stomach. If the gastric contents do not run out as soon as the outer end of the tube is lowered, cause the patient to compress the abdomen and cough or retch, when it will cause the contents to run out.

Examination of Stomach Contents.—Observe the quantity, the consistency, color, amount of mucus, and odor. Note any fragments of former meals, if such be present, or fragments of gastric membrane or blood. Fragments of membrane should invariably be examined under the microscope, for evidences of organic disease. Filter the stomach-fluid, and preserve the solid portion for further examination. The following chemical tests should be carried out on the clear filtrate as soon as possible. The following order of tests will be the most convenient:

1. Reaction.
2. Qualitative test for free HCl.
3. Estimation of total acidity.
4. Estimation of free HCl, combined HCl, and acid salts.
5. Presence of lactic acid.
6. Total organic acids.
7. Presence of pepsin and its activity.
8. Presence of rennin or milk-curdling ferment.
9. Presence of starch, dextrin, and maltose.
10. Presence of proteids and their nature.
11. Microscopical examination of the sediment.

Reaction.—The normal reaction of gastric juice is decidedly acid, and the acidity is due to HCl, acid salts (H_2NaPO_4), and occasionally organic acids, or carbonic acid. Lactic acid is usually present after an ordinary meal, but after the oatmeal test-breakfast it is usually absent, except in carcinoma. There are two sources of lactic acid—one from fermentation of the contents, especially liable to occur when the motor function of the stomach is deficient and the food is retarded longer than usual; the other, sarcolactic acid, introduced with meats and other articles of diet. Butyric and acetic acids are occasionally found in the stomach contents, due to these fermentations. The reaction of the fluid is determined with litmus paper. Unsized paper stained with other coloring matters may be used. The following table gives the coloring matters and reagents most frequently used in the examination of gastric fluid, with indications:

NAME OF COLOR.	SOLVENT.	COLOR WITH ACIDS.	COLOR WITH ALKALIES.	REACTS WITH:	DEGREE OF SENSITIVENESS.
Phenolphthalein, . . .	Alcohol, 1 per cent.	Colorless.	Pink or Red.	All acids.	Very sensitive.
Congo-red, ...	Water, 1 per cent.	Blue.	Wine Red.	Free acids only.	HCl=0.1 in 1000. Lactic=0.2 in 1000.
Tropeolin, ...	Water.	Deep Red.	Yellow.	All acids.	HCl=0.3 in 1000.
Litmus, ...	Water.	Red.	Blue.	All acids.	Very sensitive.
Dimethyl-amido-azobenzol, .	Alcohol, 0.5 per cent.	Cherry Red.	Yellow.	Free mineral acids.	HCl=0.02 in 1000.
Alizarin-sulphonate of sodium,	Water, 1 per cent.	Yellow.	Violet.	Free acids and acid salts.	. .
Boas' Resorcin Solution, . . [Resorcin, 5 gm.; cane-sugar, 3 gm.; alcohol (95 per cent.), 100 c.c.]	Alcohol.	Pink.	Colorless.	Free mineral acids.	HCl=0.05 in 1000.
Gunzburg's Solution, ... [Phloroglucin, 2 gm.; vanillin, 1 gm.; alcohol (95 per cent.), 100 c.c.]	Alcohol.	Pink.	Light Brown.	Free mineral acids.	HCl=0.05 in 1000.

These coloring matters, when used to indicate the reaction of a liquid, are called **indicators.**

Some of them, as litmus, congo-red, dimethyl-amido-azobenzol, and benzopurpurin, are frequently used in the form of strips of filter-paper colored with the dye and dried. Litmus paper is reddened by all acids and acid salts. Congo-paper turns blue in presence of all free acids, and dimethyl-amido-azobenzol is reddened by free mineral acids only. By testing the filtered gastric contents successively with paper colored with these three colors, we may determine whether it be acid, with litmus; whether the acidity be from a free acid, with congo-red;

and whether from free HCl, with azobenzol-paper. When the congo-paper is blued by the solution, showing free acid, and the paper is warmed gently over a flame, the blue color is discharged when it is due to organic acids alone, but remains blue if it has been produced by hydrochloric acid. We may confirm the presence of HCl, if it is thought necessary, by the solution of Boas or that of Gunzburg. These solutions are used as follows:

A few drops of the solution are spread out in a thin layer upon a porcelain dish or slab, which is then gently heated, and a drop of the solution to be tested is allowed to flow across it, or a glass rod dipped in the solution is drawn across the plate. If free hydrochloric acid be present, a deep scarlet-red streak is developed. If the acid be very weak, no change is observed until the solution evaporates entirely to dryness: blowing on the dish will cause the red streak to appear more rapidly. With this test it is unnecessary even to filter the gastric contents. This test is not simulated by the proteids, nor is it interfered with by acid salts, if present in the usual proportions, nor by organic acids. Its delicacy is such that it shows 1 part of HCl in 20,000 parts of water. Gunzburg's solution undergoes deterioration on exposure to light, and should be kept in a dark place or in a black bottle, and frequently renewed. For this reason the resorcin solution is to be preferred.

Detection of the Organic Acids.—There are two lactic acids met with in the stomach—sarcolactic acid and fermentation lactic acid. Sarcolactic acid is formed in the stomach from meat taken as food; the other lactic acid may be formed by fermentation. A very simple and rapid test for clinical purposes is that of Uffelmann. Diluted solutions of ferric chloride turn canary-yellow in the presence of lactic acid. A still better solution is the following:

A few drops of a dilute neutral solution of ferric chloride are mixed with two drops of pure carbolic acid, and water added until the solution assumes a beautiful amethyst-blue color. A few drops of a 1 : 2000 solution of lactic acid instantly changes the color to yellow. The delicacy of the reaction is very great. Lactates, as well as free lactic acid, unfortunately, produce this yellow color. It is said that alcohol, sugar, and certain salts can produce the same color. The test becomes certain if we first extract the organic acid from the gastric contents with ether, and apply the test to the residue left on evaporating off the ether. Acetic and butyric acids can usually be detected by the odor. The odor of acetic acid is usually unmistakable. It may also be detected by the use of ferric chloride, with which it gives a red color. Butyric acid changes Uffelmann's reagent to a tawny, reddish color, and this only occurs when it is present in over 5 parts per 1000. Alcohol is found only in rare cases of yeast fermentation.

Total Acidity.—The acidity of the gastric contents during digestion is made up of free HCl, free organic acids, acid-albumins, consisting of a loose combination of HCl and organic acids with the proteids of the food, and acid salts, chiefly acid sodium phosphate, NaH_2PO_4.

The **quantitative estimation** of the total acidity is best accomplished as follows:

To 10 c.c. of the filtered fluid, accurately measured into a beaker, three drops of a one per cent. solution of phenolphthalein is added, and enough $\frac{N}{10}$NaOH solution, accurately measured from a burette, to produce a permanent pink color. After the addition of a few cubic centimeters of the decinormal soda solution, a light rose color appears, which should not be mistaken for the end reaction. The final change of color is produced by a single drop of the alkali, and hence the addition should be made drop by drop near the end. Near the completion of the test, each drop will produce a pink-red cloud as it falls into the liquid, which will disappear on gently mixing the contents of the beaker by a rotary motion.

Estimation of Free Hydrochloric Acid.—Many methods have been devised for the estimation of the free hydrochloric acid, some of which are complicated and troublesome, while others are comparatively simple and require little skill. **Simplicity of manipulation, reasonable accuracy of results, and a small consumption of time are prerequisites of a good clinical method.**

We shall omit complicated and tedious methods. Töpfer's method of estimating free and combined HCl is simple and easy enough to be adopted as the best clinical method. It requires three separate titrations, practised upon three portions of 10 c.c. each, using three different indicators. Three portions of 10 c.c. are accurately measured into three small beakers. In No. 1 phenolphthalein is used as an indicator, and the end reaction gives the total acidity, as described above. In No. 2 dimethyl-amido-azobenzol is used as the indicator, which reacts only with free HCl. In No. 3 alizarin-sulphonate of sodium is used as the indicator, which reacts with all the elements of the acidity except acid albuminates. The total acidity is estimated in No. 1, the free HCl in No. 2; the combined HCl is found by deducting the number of c.c. used in No. 3 from that used in No. 1; and the organic acids and acid salts together are found by deducting the sum of the free and combined HCl from the result of the titration of No. 1.

The details of the above method are as follows:

The estimation of total acidity is described above (p. 595).

To the second portion of 10 c.c. three or four drops of a 0.5 per cent. alcoholic solution of dimethyl-amido-azobenzol are added, and decinormal NaOH solution is run in from a burette until the color changes from red to a clear yellow. This color is very sensitive to mineral acids, and is not affected by combined HCl, acid salts, or by organic acids, unless the proportion of lactic acid reaches 0.2 per cent. or above.

When the estimation of organic acids to be described below requires the addition of three or more c.c. of decinormal NaOH solution for 10 c.c. of the fluid, it will be best to confirm the above by a titration with the resorcin solution of Boas, or the phloroglucin solution of Gunzberg.

The number of c.c. of decinormal NaOH solution used, multiplied by 0.00365, the weight of HCl neutralized by 1 c.c., gives the weight of free HCl in 10 c.c. This, multiplied by 10, gives the weight in 100 c.c. Or, the number of c.c. of NaOH solution multiplied by 10 gives the number of c.c. required by 100 c.c. of the filtrate.

The reagents of Boas and Gunzberg give the same indications, and are used in the same way:

To 10 c.c. of the filtrate from the gastric contents add $\frac{N}{10}$ NaOH until a drop of the solution, removed on the end of a glass rod and evaporated on a white surface with the resorcin solution, fails to give a pink color. A piece of white "milk glass" answers very well for this purpose. A few drops of the indicator are spread over the glass and dried at a gentle heat. In performing the titration, a drop of the solution is removed on a glass rod and drawn across the plate, and the plate warmed over a naked flame. Blowing upon the streak as it evaporates hastens the appearance of the color.

Estimation of HCl in Combination with Proteids.—The first effect of gastric digestion upon the proteids is the formation of a small amount of acid-albumin. When the amount of HCl secreted is small, the proteids may combine with it and leave no free HCl. As long as enough of the acid is secreted to satisfy the affinities of the proteids and carry on this preliminary step in the digestion, the digestion may proceed in a fairly normal manner, even when no free HCl can be detected.

It is important, therefore, to determine the amount of this **combined HCl,** as well as the **free HCl.** This is especially important in cases of diminished HCl, **hypochlorhydria,** or total lack of free acids, **anacidity.** The combined HCl may be estimated by means of sodium alizarin-sulphonate as an indicator.

The third portion of 10 c.c. is then colored with three drops of a 1 per cent. aqueous solution of alizarin, and titrated with $\frac{N}{10}$ NaOH until a clear reddish-violet color is reached. This is reached when the free HCl, organic acids, and acid salts have been neutralized. The difference between the number of c.c. used in this titration and that used in estimation of the total acidity with phenolphthalein, gives the number of c.c. of $\frac{N}{10}$ NaOH used in neutralizing the combined HCl.

Owing to the difficulty of an inexperienced eye, in detecting the correct shade of violet in this titration, Töpfer recommends that a one per cent. solution of Na_2CO_3 be colored with the sodium alizarin-sulphonate solution, and this used for comparison, the color of this solution being imitated in the titration. The neutral tint is more nearly obtained in a one per cent. solution of sodium phosphate.

The above methods, after a little experience, give reliable results for clinical purposes, and require little skill in the manipulation.

The **estimation of the free and combined acids and acid salts** may be made by Leo's process. This process depends upon the fact that when pure precipitated chalk, $CaCO_3$, is added to the fluid, the free acids and acids combined with albumin are neutralized by the $CaCO_3$. The acidity remaining after this treatment is due to acid salts, and they may be estimated by this method. By removing the organic acids with ether and then applying the method, the free and combined HCl may be estimated.

Method.—Ten c.c. of the filtered gastric contents are shaken in a separating funnel with 50 c.c. of ether, which removes the organic acids; the liquid is then separated from the ether, returned to the separating funnel, and again shaken with 25 c.c. of ether and again separated from the ether. The fluid is then treated with 0.5 c.c. of a twenty per cent. solution of $CaCl_2$, and titrated with $\frac{N}{10}$ NaOH, using phenolphthalein as the indicator. The result gives the acidity due to acid salts plus the free and combined HCl. Another portion of 15 c.c. of the fluid is treated with about 1 gm. of $CaCO_3$, and filtered through a dry filter. To 10 c.c. of the filtrate add 5 c.c. of the $CaCl_2$ solution and titrate with $\frac{N}{10}$ NaOH with phenolphthalein. The result gives the acidity due to acid salts, which, deducted from the result obtained in the first titration, gives the free and combined hydrochloric acid.

The hydrochloric acid is greatly diminished or entirely absent in the acute stage of all fevers; in chronic gastric catarrh with atrophy of the gastric glands and amyloid degeneration of the membrane; in all cachectic states, chlorosis, certain nervous troubles, many forms of poisoning, Addison's disease; in cancer of the stomach, if it involves a considerable area or is attended with catarrh of the mucous membrane, which is usually the case. It is absent, as a rule, in cancer of the stomach, and this fact is a valuable aid in the early diagnosis of this disease.

The Organic Acids.—The presence of a considerable amount of organic acids in the stomach contents after the usual test-meal is to be regarded as pathological. The acids present may consist of lactic, butyric, or acetic acids. Butyric acid can usually be detected by an odor like that of rancid butter. Acetic acid will be detected by the odor of vinegar, especially on warming the fluid. The detection of the organic acids is more certain when they are separated from the fluid by shaking them out with ether, allowing the ether to evaporate and applying the tests to the residue. As lactic acid is likely to occur in larger amounts and more frequently than the others, several tests have been proposed for its detection. Any pronounced quantity of organic acids is generally regarded as an evidence of pyloric stenosis, deficient HCl secretion, deficient motor power, or dilatation of the stomach—in other words, of an unusual delay of food in the stomach, with resulting fermentations. When we desire to make a careful clinical test for lactic acid, it is best to give a test-breakfast of oatmeal or barley-gruel, in preference to Ewald's test-breakfast, as the former contains no lactates, while the latter may.

Uffelmann's test for lactic acid is conducted as follows:

Five drops of a strong carbolic acid solution are added to 20 c.c. of water, well mixed, and two drops of ferric chloride solution added, or enough to give a clear amethyst-colored solution. This solution changes to a canary-yellow color, with but traces of lactic acid, or with gastric fluid containing it.

Quantitative Estimation of Organic Acids.—There are a number of such methods, but the most satisfactory is that of Hehner and Seemann, sometimes called Braun's method: Exactly neutralize 10 c.c. of the fluid with $\frac{N}{10}$ NaOH, and evaporate to dryness on a water-bath, in a platinum or porcelain basin. When dry, the basin is heated over the lamp as long as the residue burns with a flame. The residue, after cooling, is extracted with boiling distilled water, filtered, and the filtrate titrated with decinormal HCl. The titration is best done by adding a measured excess of the $\frac{N}{10}$ HCl, the solution boiled to expel the carbon dioxide, and the excess of acid determined with $\frac{N}{10}$ NaOH, using phenolphthalein as the indicator. The difference between the number of c.c. of acid and alkali used will give the acidity due to organic acids present in the 10 c.c. of liquid taken. The organic salts of sodium formed during the neutralizing of the liquid are changed by the ignition of the dry residue into sodium carbonate. The amount of sodium carbonate present in the residue which is estimated in this titration, is the measure of the amount of the organic salts formed. The above method leaves little to be desired as to accuracy and simplicity.

The Volatile Fatty Acids.—When it is desired to know the quantity of these acids present, they may be estimated as follows: Ten c.c. of the filtered gastric contents are evaporated on a water-bath to a syrup, made up again to about the original volume with pure water, and the acidity determined with $\frac{N}{10}$ NaOH, using phenolphthalein as the indicator. The difference between the acidity here determined and the total acidity gives the volatile fatty acids. When lactic acid is known to be present by a qualitative test, the difference between the total organic acidity and the acidity due to volatile fatty acids may be taken to approximately represent the acidity due to lactic acid.

The Ferments—Pepsin.—The test for pepsin is accomplished by the addition of coagulated egg-albumin to the filtered gastric contents, and keeping this mixture at a temperature of about 40° C. (104° F.) for a definite time, and noting whether the albumin is corroded. If the gastric contents have been found by the above tests to be deficient in HCl, enough should be added to bring the quantity up to about two parts per thousand. In the absence of free HCl, pepsin may be absent, but there may be pepsinogen, which only needs the addition of HCl to develop the pepsin. It is best to make two tests in such cases—one of the original fluid, and another after adding two drops of diluted HCl (U. S. P.). Coagulated albumin discs, made by cutting the white of boiled eggs in thin flakes of uniform thickness and punching them out by means of a cork-borer or glass tube, and preserving in glycerin, are used for this purpose. The presence of **rennin** is best shown by carefully neutralizing 5 c.c. of the filtered gastric contents, and mixing this solution with an equal volume of carefully neutralized milk. If rennin be present, the milk is coagulated in fifteen minutes.

Digestion of Starch.—In normal digestion there is no free acidity after the usual test-meal for twenty to forty minutes. The salivary diastase continues its activity during this time, converting the starch into dextrin and maltose. At the end of one hour the greater part of the starch should be converted, and iodine should give no blue color. A reddish-violet color of a watery solution of iodine shows the presence of erythrodextrin. A blue or purple reaction with iodine, in a fluid that has been in the stomach one hour, indicates faulty amylolysis, due either to decreased diastase in the saliva, decreased secretion of saliva, or excessive acidity of gastric contents.

Digestion of Proteids.—For a description of the changes produced in proteids during gastric digestion, the student is referred to part IV, under Albumoses and Peptones.

The following tests may be employed to show the progress of proteid digestion:

Native proteids, albumin, and **globulin** are precipitated by boiling the slightly acidulated solution.

Acid-albumin, or syntonin, is precipitated by carefully neutralizing the solution with decinormal sodium hydroxide. It is best to use phenolphthalein to show the neutrality, as the solution must be exactly neutral to precipitate the acid-albumin.

Primary albumoses may be precipitated from the solution, from which the native proteids and acid-albumin have been separated, as above, by saturating the solution with $MgSO_4$ or NaCl.

Secondary albumose (deutero-albumose) may be precipitated from the filtrate from the primary albumoses, as above, by saturation with crystals of $(NH_4)_2SO_4$.

To **test for peptones,** saturate a portion of the original fluid with $(NH_4)_2SO_4$, heat nearly to boiling, and filter. To the filtrate, when cold, add phosphotungstic acid, which precipitates peptone; or apply the biuret reaction. Add to fluid an excess of NaOH or KOH and two drops of $CuSO_4$ solution. A rose-colored solution indicates peptone.

The rapidity of absorption from the stomach is tested by giving the person a capsule containing 0.2 gm. (3 grs.) of potassium iodide, and then testing the saliva at the end of each minute for the iodine reaction. **With a normal stomach, iodine appears in the saliva ten to fifteen minutes after giving the capsule.**

The iodine is detected in the saliva as follows: Strips of filter-paper are soaked in starch-mucilage and dried. One of these papers is pressed upon the tongue, removed, and then touched with a glass rod previously dipped in some yellow nitric (nitrous) acid. The appearance of a blue spot shows the presence of iodine. Some prefer to give other substances than potassium iodide. Some use a solution of common salt of known strength, drawing out what is left in the stomach after ten minutes, and estimating the salt left in the stomach. When the capsule is administered to an empty stomach, a delay in the appearance of the iodine reaction in the saliva to twenty minutes, or longer, indicates some serious organic disease of the gastric mucosa.

The motility or **motor function** of the stomach is a matter of importance, and its determination is sometimes required. When the motility is normal or increased, the food, even if it is not digested, is passed on into the duodenum before any disturbance arises from lack of digestion. The motor function may in this way compensate for lack of digestive power in the stomach. The lack of proper motor power, on the other hand, may produce dyspeptic symptoms where the digestive power is normal, because of the long delay of the food in the stomach, with secondary fermentations. Fleischer determines the motility of the stomach by giving a gelatin capsule containing 0.1 gm. (1½ grs.) of iodoform, which drug is decomposed in the duodenum, and iodide of sodium formed, which is absorbed. Iodine can be detected in the saliva in from fifty-five to one hundred minutes when administered after the usual test-breakfast.

Ewald's salol test is another chemical test of the motility of the stomach. Salol is nearly insoluble in the gastric juice, and does not enter the circulation until it is decomposed. It does not decompose until it reaches the duodenum, when it splits up into phenol and salicylic acid. The latter is then absorbed, and appears in the urine from sixty to seventy-five minutes after taking about 0.6 to 1 gm. in a capsule. Or, we may note how long the salicylic acid continues to be eliminated by the urine. If the reaction shows the acid in the urine at thirty hours or longer, it may be regarded as proof of deficient motility of the stomach. Salicylic acid is easily detected in the urine by wetting a piece of filter-paper with this fluid and dropping on the moistened spot a drop of a ten per cent. solution of ferric chloride. The edge of the drop will assume a violet color in presence of traces of salicylic acid. These papers may be dried and preserved, if necessary, as records of the test. Klemperer pours 100 c.c. (3⅓ ozs.) of pure olive oil into the empty stomach, and removes with the stomach-tube what remains after two hours, to determine how much has been passed into the duodenum. There are several mechanical methods of testing the gastric peristalsis. They depend upon the pressure the stomach exerts upon an intragastric rubber bag attached to a manometer or other recording apparatus.

Examination of the Sediment or Solid Particles Obtained from the Stomach.—The quantity, character, and appearance of the insoluble portions of gastric contents are important. We may in this way find particles of food eaten twelve to twenty-four hours before, showing a probable dilatation of the stomach or stenosis of the pylorus. Excess of the starchy elements of the food indicates an excess of gastric acidity, or a deficiency in the activity of the saliva; while an absence of bread or starch and the presence of proteid articles of diet indicate deficient acid. These indications will, of course, be more marked after an ordinary meal than after the usual test-meal. An examination of vomited matters will often reveal the above indications. The presence of mucus, in excessive amount, will be evident to the eye by its stringy, tenacious character. We may demonstrate its presence, chemically, by shaking the sediment with a weak solution of NaOH, filtering, and acidifying the filtrate with acetic acid, when the mucus will be precipitated. Excess of mucus is found in gastritis and gastric catarrh. Biliary coloring matters are usually evident to the naked eye, or they may be demonstrated by Gmelin's test (see chapter on Urine), or with the spectroscope. Blood may be detected with the unaided eye when present in considerable quantity. When in small quantity, we may use the spectroscope or the guaiac and turpentine test, mentioned under Urine. The microscope, however, will usually render these tests unnecessary.

Microscopical examination should always be made of the sediment obtained from the stomach. Besides fragments of undigested food, we may recognize the staining of these particles by bile, the presence of yeast-cells, blood-corpuscles, sarcinæ, and bacteria of various kinds. It is best to first examine the sediment unstained, and then stained with Bismarck brown or Lugol's solution, and then with methyl-blue, which last color will reveal the bacteria to the best advantage. Bacteria of various kinds are always to be found in the stomach, and are only indicative of disease when present in very large numbers. An unusually long non-motile bacillus, first recognized by Oppler, is found in most cases of gastric carcinoma. It has the power of rapidly changing sugars to lactic acid, and in carcinoma of the stomach the number of bacilli increases with this acid. Its absence in gastric disease is a very strong argument against a diagnosis of cancer. Excessive bacterial fermentations in the stomach usually give rise to dyspeptic symptoms, such as distention, pain, delayed or difficult digestion, disturbed intestinal peristalsis, toxic absorption, etc.

The Pancreatic Fluid.—The pancreatic fluid is the secretion of the pancreas poured out into the duodenum at the junction of the middle and lower third of the duodenum, through the pancreatic duct. In some lower animals there are two ducts. The rabbit, dog, and cat each have two ducts. The pancreatic juice is a transparent, colorless, odorless, saltish, alkaline fluid, effervescing with the acids. It is composed of water 90 per cent. and of solids 9.93 per cent. There are few cells, if any, in suspension, and it is, therefore, clear.

The pancreatic fluid has at least three ferments, or zymogens that furnish at least three ferments—the diastasic ferment, **amylopsin,** allied to, if not identical with, ptyalin ; **tripsin,** a peptone-forming ferment ; **pialin,** a saponifying ferment ; and **rennin.**

The secretion of the pancreatic juice begins as soon as food is taken, increases for two hours, then falls, and a second rise takes place in from five to six hours, when it falls again until about the eighteenth hour, if no more food be taken. The most marked characteristic of the pancreatic secretion is that it acts in neutral or alkaline solutions better than in acid solutions. A slight excess of HCl destroys it entirely. Its most marked action is upon proteids and starches, converting the former into peptone, with certain side-products, and the latter into maltose, which is afterward rapidly changed into dextrose. Pancreatic digestion differs from peptic digestion in certain well-marked features, which are shown below :

Active Pepsin (Acid Medium).	Pancreatin (Alkaline Medium).
Fibrin swells before dissolving.	Fibrin corroded away.
Assisted by HCl.	Assisted by Na_2CO_3.
Destroyed by Na_2CO_3.	Destroyed by HCl.
Forms syntonin, albumose, and peptones.	Forms alkali-albumin, antipeptone, hemipeptone, leucin, tyrosin, hypoxanthin, and aspartic acid.
Acts best on albumin.	Acts best on fibrin.

The pancreatic fluid is the chief digestive fluid concerned in the complete digestion of starch. It contains the ferment **amylopsin,** or **pancreatic diastase,** which readily converts starch into maltose, and finally into dextrose. The extract of the pancreatic gland is exceedingly active in its effect upon cooked starch. This secretion in the new-born is destitute of amylopsin, which makes its first appearance at about the end of the second month. Previous to this time, infants can not usually digest starch. Some infants, however, seem to be endowed with the property of digesting starch, and it would seem, therefore, that they must secrete some diastase, either in the saliva or the pancreatic fluid. The milk-curdling property of the pancreatic fluid is less marked than in the case of the gastric fluid. Of the fat-splitting ferment little is known, except that a special ferment exists in the pancreatic juice which has this property. It decomposes lecithin into neurin, and saponifies fats to a slight extent only, but sufficiently to assist in the formation of a perfect emulsion. A small amount of free acid in the oil greatly assists its emulsification with a diluted solution of sodium carbonate. For this reason a slightly rancid oil emulsifies more readily than a neutral oil.

The succus entericus is the digestive fluid secreted by the glands of the intestinal mucous membrane, principally by Lieberkuhn's and Brunner's glands. The secretion from Brunner's glands contains proteids, mucin, and ferments. It dissolves proteids, slowly changes starch into sugar, changes maltose into dextrose, and inverts cane-sugar. It is a light-yellow, opalescent, strongly alkaline fluid.

The succus entericus is most active in the dog. Its diastasic power is less than that of the saliva or the pancreatic fluid, and when it acts upon starch it forms dextrose instead of maltose. The large intestine contains none of this ferment.

The Bile.—The bile is a viscid, transparent, golden-yellow colored liquid secreted by the liver. It has a bitter taste and an alkaline reaction. Specific gravity, 1009 to 1020. Its composition is highly complex, but is represented principally by the following:

	Per Cent.		Per Cent.
Water,	91.68	Soaps,	1.39
Mucus and pigment,	0.129	Fat,	0.73
Glycocholate of sodium,	3.03	Lecithin,	0.53
Taurocholate of sodium,	0.87	Cholesterin,	0.35

The amount of bile secreted in twenty-four hours is variously stated at from 1000 to 1700 gm., or from twenty-three to forty-seven ounces. The bile is prepared in the liver from the large amount of blood received by it. More is secreted during digestion than during fasting, and the percentage of solids increases during digestion. In abstinence from food, or with a fatty diet, little is secreted; more is secreted with bread and rice, still more with meat, but most with a mixed diet. The bile is stored in the gall-bladder when not needed, and poured into the duodenum near the point of exit of the pancreatic duct. On standing, the bile becomes of a brown-yellow color and readily undergoes decomposition.

The constituents of the bile are described elsewhere.

The functions of the bile are:

First. To assist in emulsifying fats, and, in the presence of the pancreatic juice, it assists in saponifying a small portion.

Second. It readily wets the mucous membrane, dissolves fats, and it assists in their absorption.

Third. Checks putrid fermentation in the intestines.

Fourth. It precipitates the pepsin, syntonin, albumoses, and peptones from the chyme, and stimulates the production of intestinal juice.

Fifth. It contains a diastasic ferment which converts starch into sugar.

Sixth. The bile stimulates the contractions in the muscular coat of the intestines and villi, increases the moisture in the intestine, acts as a lubricant, and favors the passage of the contents of the small intestine.

A considerable portion of the bile is re-absorbed, a part of the coloring matter, at least, being excreted by the kidneys, while a part passes out with the feces. The biliary acids are mostly absorbed from the ileum and jejunum, only a trace being found in the feces.

Toxic Effects.—Obstruction to the discharge of the bile leads to its absorption by the lymphatics, giving rise to cholemia, or absorptive jaundice. The coloring matter passes into the circulation and stains the tissues yellow. The effect of the absorption of bile is to produce a slow pulse and respiration, low temperature, irritation of the skin, lassitude, headache, and coma.

THE FECES.

Chemical Composition of Feces.—An adult, upon a mixed diet, passes about 120 to 180 gm. (or 4 to 6 ozs.) of moist excrement in twenty-four hours. A vegetable diet increases the amount. If much indigestible food be taken, it may be as much as 500 gm. The consistency depends upon the amount of water present, which is usually about from 68 to 80 per cent. A pure flesh diet gives a comparatively dry feces, while substances rich in sugar yield feces with a comparatively large amount of water. The quantity of water taken has no influence upon that found in the feces. The water in the feces seems to be in direct ratio with the energy of the peristalsis.

The feces of adults have a neutral, sometimes alkaline, and rarely acid reaction. The alkalinity is derived from the ammoniacal fermentations, while the acidity is from lactic and butyric acid fermentations. Acetic and propionic acids have also been found in the feces. The copious secretion of mucus favors the occurrence of the neutral or alkaline reaction. The feces of infants fed upon milk are usually acid from the presence of lactic acid and free, fatty acids.

The color of feces depends upon the amount of altered bile-pigment mixed with them, to which the bright-yellow to dark-brown color is due, or upon the character of the food. The normal yellow-brown coloring matter of feces is hydrobilirubin. If much blood be present,—that is, after hemorrhages,—the feces are brownish-black from hematin. Preparations of iron, bismuth, or lead, taken by the mouth, color the feces black from the formation of the sulphides of these metals. Some green vegetables impart a brownish-green color, due to chlorophyll. The green color of infants' feces is believed to be due to butyric and lactic fermentation, and to the action of these acids upon the biliary coloring matters. In adults green passages may be due to a green microscopic plant, or **chlorococcus.**

The disagreeable odor of normal feces is due in great part to indol and skatol. These are products generated by the putrid bacteria, and come from the decomposition and transformation of albuminoid matters. Hydrogen sulphide, and sometimes a trace of hydrogen phosphide, ammonia and organic bases contribute to the odor of fecal matter.

The feces contain: First. Alimentary substances which are assimilable, but which have been taken in excess, as starch, fatty matters in notable quantities, and a small proportion of non-assimilable proteids.

Second. Indigestible substances, such as vegetable fiber, celluloes, chlorophyll, gums, pectic substances, resins, elastic tissue, epidermic tissue, tendons, diverse coloring matters, nuclein, chittin, insoluble salts (silicates, insoluble sulphates, ammonium-magnesium phosphate, and calcium phosphate).

Third. Products coming from the digestive tube itself; intestinal mucus, epithelial cells, biliary acids (in traces), cholesterin, and bacteria.

Fourth. Substances which failed of absorption, as emulsified fats, the free fatty acids, leucin, and bilary coloring matters.

Fifth. Decomposition products due to microbic action, as the free fatty acids from acetic up to palmitic, this last quite abundant; also butyric, isobutyric, and lactic acids, phenol, cresol, indol, skatol, stercorin, excretin, ammonium carbonate and sulphide, amins, amids, ptomaines, leucin, tyrosin, and phenylpropionic, phenylactic, and parahydroxylphenylactic acids. Some of these bodies, especially the ptomaines, acids, phenols, and coloring matters, are partly absorbed, the phenols passing into the urine in combination with sulphuric acid, as sulphuric ethers.

Sixth. Pigments. The pigments found are stercobilin, hematin, biliary pigments, hydrobilirubin, and food-coloring matters.

The following figures give the analysis of 1000 parts of fresh excrement:

	ADULT MAN.	INFANT.
Water, ...	733	851.3
Fixed solids, ...	267	148.7
Total organic matter, ...	208.75	157.1
Mineral matter, ...	10.95	13.6
Alimentary residue, ...	83	. . .

The analyses of excrement have their importance, principally, as a guide to the assimilation or malassimilation of food, and also because the excrement of various animals is employed as a fertilizer. The meconium, or the residue which accumulates in the intestinal canal during fetal life, and which is passed soon after birth, contains bilirubin and biliverdin in abundance; also biliary acids, some fatty acids, the alkaline sulphates, chlorides, and the phosphates of calcium and magnesium. It does not contain urobilin, glycogen, peptone, lactic acid, leucin, or tyrosin. Two peculiar substances are found in feces which do not occur elsewhere in the body. These are **excretin** ($C_{20}H_{36}O$) and **stercobilin, or serolin. Excretin** may be obtained from the feces by making an alcoholic extract and keeping it for a considerable time at a temperature below 0° C. (32° F.), when there is deposited an olive-colored, granular body, having a fecal odor and acid properties, from which Marcet has named it **excretolic acid.** It resembles cholesterin in its properties.

Meconium is the name given to the dark, greenish-brown, pitch-like contents of the intestine of a human fetus at birth. It is devoid of fecal odor and has an acid reaction. The microscope shows it to contain cylindrical cells from the villi, often grouped together as when covering the villi. They are usually stained green. Besides these epethelial cells, there are cholesterin plates, fat globules, and crystals of bilirubin. Meconium contains mucin, bilirubin, biliverdin, biliary acids, cholesterin, traces of fat, and fatty acids. It contains from 20 to 28 per cent. of solid matter. These feces of infants fed with normal breast-milk are lemon-yellow to orange-colored, and contain about 85 per cent. of water, 13.5 per cent. of organic and 1.5 per cent. of mineral matter. On shaking up with water, the water becomes acid, and numerous flocculi of larger or smaller size separate.

These flakes of solid matter consist of fat, fatty acids, and lactate of calcium, with usually a very small amount of casein and epithelium cells. In some cases, the fat and fatty acids will comprise as much as 50 per cent. of the total solids, although usually it is about 1.4 per cent. of the total feces. Mucin, biliary coloring matters, and micro-organisms are usually found. While the undigested casein is usually small in amount, in infants nursed at the breast, it is usually much greater in infants fed upon cows' milk. Frequently, large masses of casein and fatty acids are seen.

Changes in the Feces in Disease.—The feces of adults become a blue-clay or light gray color, when the bile is shut off from the intestine, very fetid, and contain a large quantity of fat. Occlusion of the pancreatic duct also leads to the appearance of fat in the stools. Diarrheal stools, or stools after purgative medicines, vary from the normal principally in the proportion of water, although the rapid peristalsis will usually bring down more or less undigested matters. The feces in typhoid fever are usually semifluid, and frequently contain blood and small portions of necrotic membrane, and usually contain crystals of ammonium-magnesium phosphate. They contain the Eberth bacillus, peculiar to this disease, and a poisonous base known as typhotoxin, having the formula $C_7H_{17}NO_2$, and an active toxalbumin. The Eberth bacillus decomposes cane-sugar, with the production of levorotatory lactic acid. The stools of **cholera** are thin and almost colorless, resembling rice-water. Under the microscope are seen a large number of epithelium cells from the villi, and bacterial analysis reveals the comma spirillum. Cholera stools contain indol and nitrites, and develop a blood-red color on adding dilute sulphuric acid, due to the formation of nitrate of nitroso-indol. This reaction is often used for diagnostic purposes. Poisonous bases and toxalbumins have also been found.

Dysenteric stools are characterized by an abundance of mucus tinged with blood, peculiarly fetid, and frequently containing fragments of sloughing mucous membrane. Microscopical examination sometimes reveals an ameba, known as the **ameba coli**, characteristic of certain cases of tropical dysentery. The stools of hemorrhages into the stomach, or elsewhere along the digestive tube, are dark-brown or black in color, and very fetid. **Gall-stones** are sometimes found in feces, as well as small calculi, or **enteroliths.** These bodies are best seen by thoroughly washing the feces in water, and pouring off the top liquid with the lighter floating particles, when the gall-stones or concretions, if present, will be found in the bottom of the vessel. **Animal parasites** of various kinds or their eggs, and even larvæ of insects are sometimes found in feces. Seeds of fruits, fragments of stone, metal, or other accidental substances will occasionally find their way into the feces.

Intestinal Gases.—The large and small intestines always contain more or less gas. This is usually a mixture of carbon dioxide, methane, nitrogen, and hydrogen. In strictly normal digestion, unless substances rich in sulphur are eaten in considerable quantities, sulphuretted hydrogen is not found in the gases of the colon. The CO_2 is derived in part from bacterial decomposition of carbohydrates and proteids, and in part from the blood. The CH_4 is derived from the bacterial decomposition of carbohydrates and proteids, but especially from cellulose. The nitrogen is partly derived from the blood, partly from the air swallowed, and partly from decomposition of proteids. The hydrogen is derived from fermentations of carbohydrates and proteids. Butyric fermentation of sugars generates hydrogen. The following table gives the percentage composition of the gases of the large intestine, as given by Ruge:

Vol. in 100 Vols.	Milk Diet.		Meat Diet.			Leguminous Vegetable Diet.		
	I.	II.	I.	II.	III.	I.	II.	III.
Carbon dioxide, . . .	16.8	9.9	13.6	12.4	8.4	34	38.4	21
Hydrogen, . . .	43.3	54.2	3	2.1	0.7	2.3	1.5	4
Marsh-gas, ...	0.9	. .	27.5	27.5	26.4	44.5	49.3	55.9
Nitrogen, ...	38.3	36.7	57.8	57.8	64.1	19.1	10.6	18.9

MILK.

Milk is the secretion of the mammary glands, the presence of which is characteristic of the mammalia. The milk of different animals differs somewhat in composition, but always contains the same constituents. It contains all the necessary constituents of a perfect food, and is intended for the nourishment of the young until they are able to live upon other foods. Milk is an opaque, white fluid, containing fat globules in suspension, albumin, sugar, and salts in solution, and caseinogen in partial solution. The reaction of human milk is generally feebly alkaline; that of the cow is usually neutral or faintly acid, while that of the carnivora is generally acid. Milk readily becomes acid or sour on exposure to the air, due to lactic fermentation. Microscopical examination reveals the fat in a state of perfect emulsion, the fat globules remaining suspended and separate from one another. The fluid seen between the globules is not perfectly clear, but contains small granules of caseinogen, which may be filtered out by passing it through a clay filter. The milk which is secreted for the first few days after parturition is viscid, yellow in color, of high specific gravity, and shows the presence of a few corpuscles of a peculiar character. These corpuscles seem to be epithelium cells containing fat globules, which they have not yet liberated. These cells are called **colostrum corpuscles**, and the milk of the first few days of lactation is called **colostrum.** The composition of colostrum varies in different individuals. The total solids vary from 13 to 15 per cent.; the casein, 1.5 to 3 per cent.; albumin and globulin, 1 to 7 per cent.; sugar, 3 to 6 per cent.; and salts, 0.3 to 0.5 per cent. It is believed by some that each fat corpuscle is surrounded by a shell, or membrane, of casein. Others deny the existence of this membrane.

The **specific gravity** of milk is usually determined with the hydrometer. The specific gravity of cows' milk varies from 1029 to 1035. An excess of fat lowers the specific gravity and the removal of fat raises it. The addition of water will lower the specific gravity. These facts are made use of for the detection of the ordinary adulterations of milk.

The amount of milk secreted varies with the health of the animal, the amount of food taken, and various other conditions. The amount secreted by a woman each day is about 1 liter. A good cow secretes about 7 to 10 liters. The composition of milk varies in different classes of animals with the state of nutrition of the animal, the constitution, the age, the period of lactation, and the character of the food.

Composition of Milk.—The published analyses of milk are very numerous, and the older analyses, made by older methods, differ somewhat from the analyses made by more recent methods. The comparative composition of human milk and of cows' milk is given in the following table:

ANALYSIS OF HUMAN MILK AND COWS' MILK.—(*König.*)

	Woman's Milk.			*Cows' Milk.*		
	MEAN.	MINIMUM.	MAXIMUM.	MEAN.	MINIMUM.	MAXIMUM.
Water, . . .	87.29	83.69	90.90	87.41	80.32	91.50
Total solids, .	12.71	9.10	16.31	11.59	8.50	19.68
Fat, . . .	3.78	1.71	7.60	3.66	1.15	7.09
Milk-sugar, .	6.04	4.11	7.80	4.92	3.20	5.67
Casein, . .	1.03	0.18	1.90	3.01	1.17	7.40
Albumin, . .	1.26	0.39	2.35	0.75	0.21	5.04
Albuminoids,	2.29	0.57	4.25	3.76	1.38	12.44
Ash, . . .	0.31	0.14	(?)	0.70	0.50	0.78

Besides the constituents mentioned in the table, milk also contains very small quantities of certain extractives, among which are creatin, leucin, the odoriferous principle, lecithin, nuclein, etc. It also usually contains certain gases, principally carbon dioxide, oxygen, and nitrogen. Colostrum contains a larger proportion of solid matter than ordinary milk, a larger amount of proteid materials, and less sugar.

The following table shows many differences between human and cows' milk not shown in the above table, and which are of importance in adapting the latter to infant feeding:

TABLE SHOWING DIFFERENCES BETWEEN HUMAN AND COWS' MILK.

PROPERTIES.	HUMAN MILK.	COWS' MILK.
Physical appearance.	Bluish, translucent, odorless, sweetish.	Opaque, white or yellowish-white, distinct odor, feebly sweet taste.
Specific gravity.	1026 to 1036.	1029 to 1035.
Reaction.	Amphoteric or slightly alkaline.	Amphoteric or slightly acid; becomes quickly acid on exposure to air.
Behavior on boiling.	Does not coagulate, and forms a very slight pellicle, scarcely observable.	Does not coagulate, but forms a distinct pellicle of casein and lime-salts.
Spontaneous coagulation.	Coagulates only after one to two days, at room-temperature.	Coagulates after six to twelve hours at room-temperature; due to lactic acid.
Coagulation with rennet.	Coagulates incompletely in small, isolated flocculi, never forming visible curds.	Coagulates at body temperature, separating into curdy masses and opalescent whey.
Fat.	Butter yellowish, similar to cow-butter; sp. gr. at 15° C. = 0.966; melts at 34° C.	Butter yellow-white; sp. gr. at 15° C. = 0.949 to 0.996; melts at 35.8° C.
Composition of fat.	Olein, palmitin, stearin, butyrin, caprin, caproin, myristin.	Olein, palmitin, stearin, caproin, caprylin, caprin, laurin, myristin, arachin, butyrin, lecithin, cholesterin.
Relation of acids.	Volatile acids relatively small; oleic acid, one-half non-volatile acids.	Volatile acids relatively large, oleic acid small, palmitic and stearic large.
Caseinogen.	Precipitated with difficulty by salts and acids; easily soluble in acids; leaves no pseudonuclein with peptic digestion.	Precipitated easily by salts and acids; precipitate not easily soluble in excess of acids; leaves residue of pseudonuclein.
Composition of proteids.	Lactalbumin, lactoglobulin, and casein; ratio of casein to albumin, 1 to 1.2 (?).	Lactalbumin and globulin small; casein to albumin, 1 to 7, or even 1 to 10.
Mineral matters.	Relatively small amount; contains one-sixth as much CaO and one-fourth as much P_2O_4 as cows' milk.	Contains relatively large amount.
Nuclein and lecithin.	Nuclein, 1 2 gm. per liter (Siegfried); lecithin, 1.7 gm. per liter (Stoklasa).	Nuclein, 0.6 gm. per liter (Siegfried); lecithin, 1 gm. per liter (Stoklasa).
Bacteria.	Generally sterile; rarely staphylococcus albus and aureus.	Contains numerous bacteria, and occasionally typhoid, diphtheria, tubercular organisms, etc.

The proteids which occur in milk are probably three in number—caseinogen, lactalbumin, and lactoglobulin. Lactoprotein and peptone have also been described by some observers. When milk is allowed to stand at the ordinary temperature, a part of its lactose is converted by fermentation into lactic acid. When this has accumulated to a considerable extent, the caseinogen is precipitated. When milk is treated with rennin, or gastric juice containing this ferment, the caseinogen is rapidly converted into casein, and at the same time coagulation or precipitation occurs. When rennet is added to cows' milk, the result is a coherent clot or curd and a clear, yellowish fluid called whey. The curd contains the fat entangled with the casein. The whey contains the albumin, sugar, and salts. In human milk the curd is formed of smaller flocculi, and the same appearance may be produced with cows' milk if it is previously boiled or largely diluted with lime-water. Caseinogen is often compared to alkali-albumin; the latter, however, does not coagulate with rennet, and is readily soluble in acids, while caseinogen is not. Caseinogen somewhat resembles the globulins in its behavior with neutral salts. The globulins, however, coagulate when heated, while caseinogen does not.

Casein.—This name is sometimes restricted, as above, to the proteids formed, by the action of rennet and acids, from the caseinogen of milk. Casein is the chief constituent of cheese.

Lactalbumin.—After the precipitation of the casein by acetic acid, this proteid is left in the solution. The scum which forms on the top of milk on boiling is probably formed by the coagulation of a part of the lactalbumin and caseinogen.

The boiling of milk before it is used as food is advantageous, in that all germs of disease are destroyed, and that the rennet gives a flocculent instead of the heavy, curdy precipitate. The lactalbumin is modified by the boiling, and precipitates on adding acid to the cold boiled milk.

Lactoglobulin.—Various observers have discovered other proteids than the two mentioned above. Lactoglobulin, whey proteid, lactoprotein, proteoses, peptones, and nuclein have been mentioned as occurring in milk, but our knowledge of the amount present is so slight that we will pass them with the mere mention. It is claimed by some that their supposed presence is due to faulty methods of examination. The lactoglobulin has the property of liquefying cooked starch and of partially digesting it. This property is destroyed by sterilization.

Fat.—The chemical composition of milk-fat is very nearly like that of adipose tissue, with small quantities of the triglycerides of butyric, caproic, caprylic, myristic, and arachinic acids. Milk also contains minute quantities of lecithin, cholesterin, and yellow lipochrome.

Cream is simply the upper layers of milk which has been left to stand, and in which the fat globules are more numerous than in whole milk. The amount of cream that will separate from the milk in twenty-four hours is sometimes made use of as a test for the richness in fat. A fair market milk will give from 10 to 12 per cent. of cream, while rich milk will frequently give from 15 to 20 per cent., or even more.

Butter is the fat of milk in which the fat globules are separated from the other constituents by mechanical agitation in the churn. About one-sixth of the fat remains in the buttermilk. Buttermilk contains, therefore, about from 0.5 to 1 per cent. of fat. Butter also contains small quantities of casein and lactose. Butter from human milk is richer in fluid fats than that made from cows' milk. By exposure to the air butter becomes rancid, due to the saponification of some of the glycerids of the fatty acids.

Milk-sugar, as will be seen in the above table, occurs to the extent of about 4.5 per cent. in cows' milk and about 6 per cent. in human milk. The characteristics of this sugar have been described in part v.

The **salts** of milk are the phosphates of potassium, sodium, calcium, and magnesium, with chlorides of potassium and sodium, and a trace of iron, which is in combination in the nuclein. The most of the phosphorus of human milk and about one-half that of cows' milk exist in organic combination in the nuclein and lecithin.

Sterilized and Pasteurized Milk.—Owing to the ease with which milk undergoes the lactic fermentation and becomes sour, various methods have been tried to preserve milk for use in large cities. Subjecting the milk to a low temperature has been in use for a long time, and is of great service. Milk kept at a temperature of about 10° C. (50° F.) will keep sweet for a number of days.

For use as a food for infants, this has been found to be impracticable. Even with the greatest precaution, the lactic ferment will slowly progress and will grow in such abundance, as soon as the food is taken, as to disturb the child's digestion. This may be and often is prevented by heating the fresh milk to 100° C. (212° F.) for fifteen to twenty minutes in a bottle or jar, closing it while hot, and excluding air until needed. This "**sterilized milk**" is to be found in the markets. It has not met with the success in use that was at first expected. The heat coagulates the lactalbumin, the globulin, and modifies the casein. On acidifying sterilized milk all the proteids are precipitated at once in a firm curd that is not easily digested, but appears in the feces. Experience shows that children fed upon this milk do not thrive as well as when fed on raw milk.

The nuclein and lecithin of the milk are decomposed at a temperature of 100° C. (212° F.), and this probably alters its nutritive qualities and accounts for the tendency of children to become rachitic when fed exclusively upon sterilized milk or condensed milk.

The temperature at which the above changes in the proteids begin is about 75° C. (167° F.). If milk is heated to about 68° C. (155° F.), or not above 70° C. (158° F.), many organisms are killed, and others so weakened that the keeping quality is greatly improved, without altering the taste or composition. This process is called **Pasteurization,** and is now used to preserve milk for use as food for infants.

Modified Milk.—By this term is meant cows' milk that has been changed in composition so as to resemble the composition of human milk. As the caseinogen of cows' milk is about three or four times that of human milk, and the albumin is but half that of human milk, there is a very decided difference in the digestibility of the two. Rennin coagulates the caseinogen only. The curd formed in the stomach from cows' milk is more abundant, and forms tough masses, difficult to digest, while that from human milk is slight in amount and flocculent.

The sugar of cows' milk is present in about 4.5 per cent., while that of human milk is nearly 2 per cent. higher. These and other differences in composition and behavior of the two milks, shown in the table on page 611, make it necessary to modify the composition of cows' milk for the successful nourishment of infants. Various methods have been proposed for accomplishing this, and have led to the production of numerous preparations put upon the market as baby-foods.

One of the most successful of the attempts to modify cows' milk to imitate human milk is a mixture of milk, cream, water, lime-water, and milk-sugar, made to correspond to the average analysis of human milk, and the mixture Pasteurized. Such mixtures are now to be had in many large cities from **milk laboratories,** established for the purpose of furnishing them. Milk of any desired composition may be ordered from the laboratory, and this composition may be changed at will.

The milk used for this process, as well as the cream, must be fresh, and the cream should be of nearly constant composition. The only way to secure this is to use cream separated by the centrifugal machine.

With such a cream, containing 20 per cent. of fat, the following proportions will give very nearly the composition of average human milk:

Milk,	2 parts; or,	Milk,	4 fluidounces.
Cream,	3 parts; "	Cream,	6 fluidounces.
Water,	10 parts; "	Water,	20 fluidounces.
Lime-water,	1 part; "	Lime-water,	2 fluidounces.
Milk-sugar,	½ part; "	Milk-sugar,	7 drachms.

The analysis of this mixture will give about the following results, when a good milk is used:

Water, . . .	88.42	Fat, ...	4	Sugar, ...	6.26
Solids, . . .	11.58	Proteids, . . .	1.11	Ash, ...	0.21

Such mixtures can not be regarded as very near imitations of human milk. Even when the mixtures are made at milk laboratories, certain peculiarities of human milk, shown in the comparative table on page 611, can not be imitated in this way. The fat of the two milks are of different composition, and the process of centrifugal separation of the cream partially destroys the emulsion, and collects the fat globules into small masses and renders the fat less easily digested. The different amounts of nuclein and lecithin in the two milks are unfavorable to cows' milk, as regards its nutritive qualities. It is in the relative proportion of the two proteids that the most difficulty is experienced. The above mixture contains 1.11 per cent. of proteids, of which 0.86 per cent. is caseinogen and 0.25 is albumin and globulin. Human milk usually contains slightly more albumin than caseinogen, a fact that is of great importance in the behavior of the milk with rennin or with acids—*i. e.*, in the first stages of digestion. This relation between the caseinogen and albumin determines more than any other factor the character of the curd produced on coagulation. The larger the proportion of albumin to the caseinogen, the finer the flocculi formed. The actual amount of proteids in human milk varies at different periods of lactation, being greatest during the first week. The following figures show this variation, and the relative percentage of albumin and caseinogen (Camerer):

PERIOD OF LACTATION.	TOTAL PROTEIDS.	CASEIN.	ALBUMIN AND GLOBULIN.
Eighth to eleventh day, . . .	2.53	0.91	1.62
Twentieth to fortieth day, ...	1.74	0.55	1.19
Two to three months, ...	1.46	0.46	1.00
Eight months, ...	1.19	0.35	0.84

From the above analysis we see that the amount of total proteids in human milk during the first two weeks of lactation is about 2.5 per cent., and in many cases 3 per cent.; but of these nearly two-thirds are soluble albumin and globulin instead of caseinogen. We may increase the albumin in the above-described mixture by the addition of egg-albumin in the proportion of the white of two eggs to the quart of mixture. Assuming that the white of an average egg will weigh 30 gm., or 1 oz., and that it contains 12.2 per cent. of albumin, the whites of two such eggs would contain 7.32 gm. of albumin, and would add approximately 0.75 per cent. of egg-albumin to the above mixture, and give 1.86 per cent. of proteids, of which 0.86 per cent. will consist of caseinogen and 1 per cent. will consist of albumin. While egg-albumin differs materially from lactalbumin, the physical behavior of the mixture is more nearly like that of human milk, and experience shows it to be well suited for infant nutrition.

A rational method of modifying cows' milk for use as an infant food is the following: The milk should be allowed to stand in a cool place for three or four hours, to allow the cream to separate. When the milk is received in bottles, as is the custom in large cities, this will be unnecessary. Siphon off from the bottom of the containing vessel two-thirds of the milk, leaving the cream and upper portion of milk undisturbed. This may be easily done with a small rubber tube, previously filled with water to start the siphonage. To the milk thus drawn off add a teaspoonful and a half of essence of pepsin or liquid rennet, warm to blood heat, 37° C. (98.6° F.), and keep at or near that temperature for twenty to thirty minutes, or until the curd separates. Then warm, with vigorous stirring, to 68° C. (155° F.) and filter, while hot, through muslin. This whey will contain approximately 1 per cent. of fat, 4.5 per cent. of sugar, 0.8 per cent. of soluble albumin, and 0.7 per cent. of salts. When cold, this whey is added to the rich milk left in the containing vessel.

The mixture thus obtained will contain approximately 1.2 per cent. of caseinogen, 0.8 per cent. of albumin, 4.5 per cent. of sugar, 3 per cent. of fat, and 0.7 per cent. of salts. To a quart of this mixture we must add 1.5 per cent. of milk-sugar to bring the percentage of this constituent up to 6 per cent. This will require about one-half ounce, or a heaping tablespoonful, of powdered milk-sugar. This mixture is very successful in practice. The behavior of the mixture, when coagulated with dilute acid, is strikingly like that of human milk.

Changes Produced in Milk by Disease.—The milk of a strong, healthy woman is more nourishing than that of the weak, sickly woman. The character of the secretion of milk in the human subject, as well as in some of the lower animals, is greatly varied by the emotions, and milk secreted during periods of excessive mental excitement has frequently proven poisonous to the young. Certain drugs pass through the mother into the milk, as, for example, iodine, arsenic, antimony, lead, zinc, bismuth, and mercury. Opium and morphine, although they may not be detected in the milk, have frequently passed into the milk in sufficient quantities to narcotize the infant. In the cow the character of the food and the state of the health have an important bearing on the composition of the milk. In cases of the cattle-plague the milk has been found to contain blood.

The milk in cases of tuberculosis, a common disease in cows, is capable of communicating this disease to calves, as well as to human subjects. Milk from tuberculous cows should never be used. The milk from foot-and-mouth disease is also injurious. Milk is often the carrier of the infection of contagious diseases, as measles, scarlet fever, diphtheria, small-pox, and typhoid fever.

Milk is a good cultivating medium for the growth of various bacteria, and several characteristic bacteria producing coloring matters occur in milk, one giving it a blue, another a purple-red, and another a yellow color. Milk is sometimes rendered poisonous by certain bacterial growths. These poisons are either ptomaines or toxalbumins produced by the growth of these bacteria.

The Adulterations of Milk.—The adulterations usually practised are the extraction of cream, or the addition of water, or both. Occasionally the addition of some foreign substance, as sodium carbonate, common salt, or sugar, is met with.

The detection of the adulterations of milk usually depends upon the determination of the specific gravity, the fat, total solids, and the ash. The quantity of these ingredients is not perfectly uniform, and hence certain limits of allowable variation have been determined upon from time to time. The standard adopted in many States in this country is a specific gravity not less than 1029 and total solids not less than 12 per cent., of which 3 per cent. shall be fats. The legal limits for total solids vary from 12 to 13 per cent., and the solids not fat from 8.5 to 9.5 per cent. The Society of Public Analysts of Great Britain have adopted for total solids, 11.5; fat, 3; and solids not fat, 8.5 per cent.

Milk Testing.—There is no instrument of simple construction which will with certainty detect the presence of a small amount of adulteration in milk. The **lactometer**, or **lactodensimeter**, which has been employed very largely in the sanitary inspection of milk, is a hydrometer with a scale covering the variations usually met with in milk. (See Fig. 4.) The **lactometer** of the New York Board of Health is a hydrometer on which the scale is so constructed that 100° indicate a specific gravity of 1029, the supposed lowest specific gravity of pure milk. The space between 1000, the specific gravity of water, and 1029 is divided into 100 arbitrary degrees. The scale is extended to 120°, which corresponds to a specific gravity of 1034. When taken alone, it is of very little value. If, however, it be taken with the estimation of either the total solids or the fat, it is of considerable service. In very exceptional cases the milk of a single cow may have a specific gravity below 1029, but such milk should be regarded as abnormal. Such depression of the specific gravity never occurs in the mixed milk of several well-fed cows. A specific gravity below 1029, therefore, unless accompanied by an excessive amount of fat, may be taken as evidence of contamination, probably with water.

The fat for such examinations may be estimated by the creamometer, or by some form of lactoscope, or the lactobutyrometer. The creamometer, or cream gage, is simply a graduated cylinder, the graduations being $\frac{1}{100}$ of the total capacity of the cylinder to the zero mark. (See Fig. 4.) The milk is added in the cylinder to this zero mark, and allowed to remain at rest for twenty-four hours, when the number of the divisions covered by the cream is read off. This should not be less than ten per cent. The **lactoscope** depends upon the assumption that the opacity of the milk is proportional to the amount of fat which it contains. In **Feser's lactoscope** (Fig. 5), a measured volume of milk is placed in a graduated vessel, A, by means of the pipette, B. It is then diluted with water until the black lines of the inner cylinder of opaque white glass can be seen through the layer of the mixture between the walls of the inner and outer cylinders. It is then only necessary to read off the percentage of fat on the scale of the outer cylinder at the surface of the liquid. This method of determining the fat in milk, although answering for the purpose of municipal control, is not to be depended upon for scientific purposes, or as

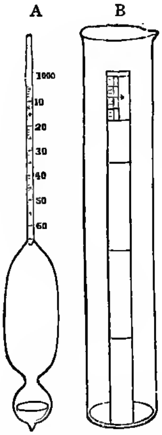

FIG. 4.—A. HYDROMETER. B. CREAMOMETER.—(*Starr.*)

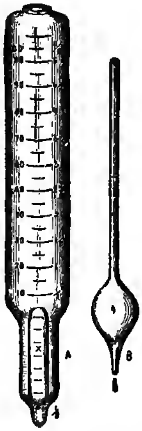

FIG. 5.—FESER'S LACTOSCOPE.—(*Queen.*)

evidence upon which to base legal proceedings. The lactoscope is of doubtful value in estimating the fat in human milk. In a large experience with this instrument the author has seldom seen the readings vary more than 0.3 per cent., in cows' milk, from the accurate methods. Usually it is much nearer than this.

The Chemical Analysis of Milk.—An easy, rapid, and satisfactory method for estimating the fat in milk is that known as the **Werner-Schmid process.** Ten c.c. of the milk are measured out into a long test-tube, holding 50 c.c. and graduated at every 10 c.c., and treated with 10 c.c. of strong HCl. If desired, the milk may be weighed into a small beaker-glass and then washed into the test-tube with the acid, when the test-tube need not be graduated. After mixing the milk and acid together, the mixture is heated to boiling, or, it is loosely corked and heated in a water-bath for five or ten minutes, or until the liquid turns brown, but not black. The tube and contents are then cooled, 10 c.c. of well-washed ether added, corked, and the mixture well shaken. As soon as the ether separates from the remainder of the fluid, the cork is removed and the wash-bottle arrangement shown in figure 6 inserted. The lower end of the exit tube is now adjusted by sliding it in the cork so that it is just above the line of separation of the two fluids. The ether solution of the fat is now blown off into a weighed beaker or flask. Two more portions of ether, of 10 c.c. each, are added, shaken up, and blown off into the first portion. The ether is now distilled off and the fat dried in a water-oven and weighed. The amount of fat so obtained represents that contained in 10 c.c. of milk, or in the amount weighed out.

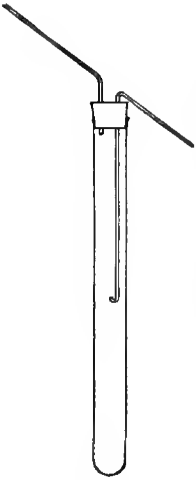

FIG. 6.

The results agree quite closely with the **Adams method,** described below.

The **total solids and water** are determined by placing in a weighed platinum dish a weighed quantity of the milk to be tested—say, about 5 gm. This is then placed upon a water-bath and evaporated to dryness. It is now transferred to the water- or air-oven, and dried at 100° C until it ceases to lose weight. The loss in weight represents the water; the residue represents the total solids. Where great accuracy is unnecessary, the fat may be determined in the residue by treating it with warm ether and pouring this through a small filter, and repeating this process until the fat is completely dissolved out, which will require usually about 75 c.c. of ether. The ether is allowed to evaporate, and the fat which remains behind is weighed; or, the residue left in the dish may be again weighed, when the difference between this weight and the weight of the dry solids will give the amount of fat. The results are about 0.3 per cent. too low.

The ash may now be determined by igniting, at a dull-red heat, the residue left after treatment with ether until all the organic matter is burned off, weighing the residue, and calculating it as ash.

The Centrifugal Method.—This method of separating the fat from milk for analytical purposes is quite satisfactory. The centrifugal machine has, in recent years, come into common use both in the chemical laboratory and in the physician's office.

In Babcock's method the milk is mixed with an equal volume of commercial sulphuric acid in a specially constructed bottle, provided with a long, narrow, graduated neck. After whirling the bottle in a centrifugal machine, the fat is separated, and the percentage is read off on the scale on the neck of the bottle. Leffman and Beam * have modified the process by adding to the milk a small quantity of a mixture of amyl-alcohol and hydrochloric acid, before adding the sulphuric acid. The size of bottle, the quantity of milk taken, and the centrifugal machine used is somewhat different from those of Babcock. More recently, this method has been modified by Koplik so as to adapt it to the small centrifugal machines used by physicians (Fig. 12). This is especially valuable in the examination of human milk. It requires little skill, is quickly done, and the results are quite satisfactory.

The necessary pipettes and bottles are shown in figure 7.

The process of making a determination of fat by this method is as follows: Fill the pipette to the mark with the milk to be examined. This represents 2.92 c.c. of milk. Introduce the end of the pipette carefully, so that the end is well into the body of the bottle, before delivering the milk. Cleanse the pipette, fill it with pure, strong sulphuric acid, and pour this into the milk in the bottle, with the same care as before. Add 0.6 c.c. of a mixture of equal parts of amyl-alcohol and pure concentrated hydrochloric acid. Mix the contents of the bottle by a rotary motion, and then fill the bottle with a freshly prepared mixture of equal parts of sulphuric acid and water. Two bottles are thus prepared and placed in the machine and rotated two or three minutes, when the fat will have separated and the percentage may be read off on the graduated neck of the bottle. The space between the smaller lines represents 0.3 per cent. of fat. To insure greater accuracy, it is best to use a small hand magnifying glass, with the aid of which ½ or even ¼ of a division may be read, and thus indicate 0.1 per cent. If the fat layer is seen to cover 8 divisions on the scale, the percentage of fat in the milk is 2.4. If it covers 10 divisions, it indicates 3 per cent. of fat. The amyl-alcohol and hydrochloric acid mixture may be kept for some weeks, but when it has become dark-colored it must be renewed. These tubes are made by Richards & Co., New York.

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* "Analysis of Milk," P. Blakiston's Son & Co., Philadelphia.

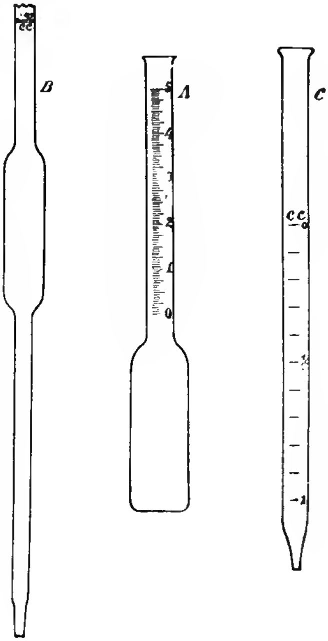

FIG. 7.

For this estimation there is needed a specially graduated bottle (*A*, Fig. 7), a pipette (*B*), delivering 2.92 c.c., for measuring the milk and the sulphuric acid, and a 1-c.c. pipette (*C*), graduated into tenths, for measuring the mixture of amyl-alcohol and hydrochloric acid. Special aluminum receivers for the bottle (*A*) are adapted to the ordinary centrifuge, such as shown in figure 6.

Calculation of Fat from Total Solids and Gravity.—This method is useful when great accuracy is not demanded, and may be used in clinical examinations of human milk. It rests upon the assumption that one per cent. of milk solids not fat raises the specific gravity by a definite amount, while one per cent. of fat lowers it by a definite amount. An accurate determination of the specific gravity and total solids will, therefore, furnish the necessary data for calculating the amount of fat. The formula by which the calculations are made is that of Hehner and Richmond. It is as follows: $F = 0.859\ T - 0.2186\ G$, in which F = fat, T = total solids, and G = specific gravity, as expressed in the third and fourth figures. Or, when the fat is known, the formula may be transposed so as to calculate the total solids, as follows:

$$T = \frac{F + 0.2186\ G}{0.859}$$

The specific gravity must be determined with care, and for rapidity only 5 c.c. of milk are evaporated to dryness to determine the total solids. This may be conducted in a watch-glass, using 2 or 3 c.c., if a platinum dish is not at hand.

To illustrate the use of these formulæ let us assume, in the examination of a given milk, that the specific gravity, or G, was found to be 1030, and that the total solids, on examination, gave 12 per cent. Substituting these figures in the above formula, we have:

$$\text{Fat} = 0.859 \times 12 - 0.2186 \times 30 = 3.75.$$

Or, if the fat has been determined instead of total solids, and found to be 3.75 per cent., then:

$$\text{Total solids} = \frac{3.75 + 0.2186 \times 30}{0.859} = 12.$$

Another method has been proposed for calculating the solids not fat, from data afforded by the lactometer, specific gravity and Feser's lactoscope, by means of the formula $\frac{G - A}{0.00375}$, where G equals the specific gravity of the milk and A the remainder obtained on multiplying the percentage of fat, as shown by the lactoscope, by 0.001 and deducting this from 1000. For example: Suppose in a given sample of milk the specific gravity, or G, is found to be 1030. The value of A in the above equation will be found by multiplying the per cent. of fat, 3 7, by 0.001, which will be equal to 0.0037, which, deducted from 1000, equals 0.997. Substituting 1030 for G, and this remainder, 0.997, for A in above equation, we have:

$$\frac{1030 - 0.997}{0.0037} = 8.9 \text{ per cent. of solids not fat.}$$

These short methods will be found useful in the examination of human milk, where long, tedious processes are not likely to be entered into. While some of them are not scientifically accurate, they are sufficiently so for clinical purposes and for the use of sanitary inspectors in sorting milks.

In the accurate estimation of the fat in milk, the officially recognized method is that of Adams. Instead of drying the solids in the usual way, the milk is absorbed by bibulous paper previously thoroughly exhausted with ether and alcohol. This paper is usually cut in the shape of long strips, and these are rolled into a coil and put in a special apparatus known as an extractor, and shown in figure 8. The coil is put into the chamber of the middle piece of the apparatus, which is then connected with a condenser, as shown. Sufficient ether to fill this chamber is put into the flask below, which is gently warmed. The ether distils up into the condenser and runs back upon the coil, filling the chamber until it flows over through the siphon-tube into the flask below. This is repeated until exhaustion is complete. The ether is finally distilled off, and the fat in the flask is dried and weighed. The results obtained by this method are about 0.3 per cent. higher than those obtained by the method above described, of evaporating in a platinum dish and treating with ether. This has been adopted by official chemists, both in England and in this country, as the standard method of estimating fat. For the accurate estimation of fat in milk in well-equipped laboratories, it leaves little to be desired in the way of accuracy, but is difficult without these facilities.

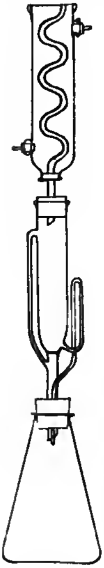
FIG. 8.

Milk Standards.—For ordinary purposes the estimation of the total solids, the fat, and the ash are considered sufficient to determine the question of the adulterations usually met with in the market. The standards that have been fixed by law in a number of the States all refer to specific gravity, fat, and total solids. Prosecutions are, therefore, usually based on these data. To calculate the percentage of pure milk in a mixture, the following formula may be adopted, based upon the legal standard of the State of New York—viz., 12 per cent. of milk solids, 3 per cent. of fat, and 9 per cent. of solids not fat:

$$9 : \text{solids not fat} :: 100 : x = \text{milk used in making 100 parts of the mixture.}$$

For other standards the first member of the equation will be the legal percentage of solids not fat. When the solids not fat are less than 9 per cent., it indicates some form of falsification. Suppose, for example, the solids not fat in any given analysis were 8.1. Substituting this in the above proportion, we have :

$$9 : 8.1 : : 100 : x = 90;$$

or, this sample of milk had been made from 90 per cent. of milk and 10 per cent. of water. If the milk is skimmed, the percentage of fat removed can be ascertained by the following formula:

$$\tfrac{3}{9} \times S - F = x;$$

in which S = solids not fat, and F = fat found. Suppose, for example, the fat in a given case be 2 per cent. and the solids not fat 8 per cent. Substituting these in the above equation, we have :

$$\tfrac{3}{9} \times 8 - 2 = x = 2;$$

that is, 2 per cent. of fat has been removed from this milk.

The Estimation of Sugar.—For clinical purposes a sufficiently correct estimation of milk-sugar can be made by exhausting the residue that remains after the extraction of the fat from the dry solids with ether with weak boiling alcohol. This dissolves the sugar and the soluble portion of the ash. The solution is filtered, evaporated to dryness in a platinum or a porcelain capsule, and weighed. The residue is then ignited and the ash weighed and deducted from the weight of sugar and ash, to obtain the amount of sugar.

Lactose may also be estimated with Fehling's solution, after coagulation with acetic acid and removal of the casein.

The Determination of Casein.—Casein and albumin are generally determined by difference. When the direct determination is desired, they may be precipitated by tannin, filtered, the precipitate dried, and washed with a mixture of one part of alcohol to three of ether until the washings show no trace of tannin. The residue is then dried and weighed.

The albuminoids can also be determined by the method of Ritthausen, who employs a solution of $CuSO_4$, containing 6.5 gm. to the liter, and a solution of alkali of the strength of 14.2 gm. of KOH or 10.2 gm. of NaOH to the liter. The copper salt precipitates the albuminoids, together with the fat. Twenty c.c. of milk are taken, and diluted with water to 400 c.c. ; 10 c.c. of the copper solution are then added, with constant stirring, until the coagulum settles and the supernatant liquid is clear. The alkali solution is now added until the liquid is neutral, and the contents of the beaker are filtered, using a previously dried and weighed filter-paper. The precipitate is all transferred to the filter. It is washed first with water, then with diluted alcohol, and finally with ether, until all fat is removed. The remaining precipitate is again washed with alcohol and dried at 110° C. (230° F.) and weighed. The bluish mass is burned, and the loss, after deducting the weight of the filter-paper, is reckoned as casein and albumin.

Estimation of Caseinogen and Albumin.—In the examination of human milk, it is a matter of great importance to know the proportion between the albumin and caseinogen, because of the great importance of this ratio in infant nutrition. A rapid and easy method of separating these proteids for analytical purposes is very desirable, and several processes have been proposed. The most of them require too much time for clinical purposes. Berggran and Winkler have proposed a volumetric method based upon the fact that the proteids of milk form insoluble compounds with a solution of potassium-mercuric iodide containing ferric chloride, containing a fixed and known amount of free iodine.

The solutions used are:

1. A solution of 0.2 gm. of mercuric iodide dissolved in 10 c.c. of a 10 per cent. solution of potassium iodide. To this is added 1 c.c. of a 10 per cent. solution of ferric chloride, and the whole made up to 100 c.c. with water. This solution should be made fresh each twenty-four hours, as it does not keep well. The potassium-mercuric iodide and ferric chloride may be kept in two separate solutions, which may be mixed when required.

2. A $\frac{1}{100}$ normal solution of sodium thiosulphate, made by dissolving 2.476 gm. of selected crystals in a liter of water. Each c.c. of this solution should exactly decolorize 1 c.c. of solution No. 1 in the presence of a few drops of starch solution.

3. A solution of starch made by boiling 1 gm. of starch in 100 c.c. of water and filtering. The milk to be examined is allowed to stand until the cream has separated, or the most of the cream is separated with the centrifugal machine.

Five c.c. of the skimmed milk are diluted with 20 c.c. of water, and to this exactly 5 c.c. of solution No. 1 are added. After standing five minutes, a few drops of solution No. 3 are added, and the solution titrated with solution No. 2 until the blue color is discharged. If the relation between solutions Nos. 1 and 2 is known, it is easy to determine how many cubic centimeters of solution No. 1 have been used by the proteids.

Suppose, for example, 5 c.c. of skimmed milk be treated with 5 c.c. of solution No. 1, and the titration with solution No. 2 required 0.4 c.c.

Suppose a previous titration of 5 c.c. of solution No. 1 required 5.3 c.c. of solution No. 2. One c.c. of No. 2 then corresponds to $5 \div 5.3 = 0.943$ c.c. of solution No. 1, and to 0.000943 gm. iodine. The back titration in the above example required 0.4 c.c. of solution No. 2, and therefore there was the difference between 5.3 and 0.4 c.c. used by the proteid in the 5 c.c. of milk, or 4.9 c.c.

$4.9 \times 0.000943 = 0.00462 = 0.00462$ gm. iodine, combined with the proteids.

By a large number of analyses, it has been found that 1 gm. of iodine is precipitated by 16 gm. of milk-proteids, and therefore by multiplying the iodine used by 320, the proteids in 100 c.c. of milk are found. In the above example:

$0.0046 \times 320 = 1.472$ gm. proteid in 100 c.c. of milk.

To estimate the albumin, 10 c.c. of milk are put into a 50 c.c. flask or cylinder, diluted, warmed on a water-bath to 40° C., and a few drops of acetic acid added and made up to the mark. The liquid is filtered, 25 c.c. of filtrate taken (5 c.c. of milk), and treated exactly as above. The difference between the total proteids and the albumin obtained by the second titration will represent the caseinogen.

Detection of Impure Water.—The addition of water to milk, if it be pure water, can be regarded as harmless to adults. It is rather a sophistication than a harmful adulteration. As this is usually well-water, which may itself be impure, it becomes a matter of importance, because the water may carry with it germs of typhoid fever, cholera, or other diseases, and will impart to the milk infectious properties. To detect impure water in milk the following process may be used: The milk is coagulated with acetic acid and filtered. To a suitable quantity of the whey add equal parts of a solution of naphthylamin sulphate and a freshly prepared solution of sulphanylic acid in sulphuric acid. The test may be made in an ordinary test-tube or in a cylinder. If the milk contains nitrites, due to an impure water, a rose-red color will appear, varying in intensity with the amount of nitrites present, and deepening on standing. The test is very delicate (p. 147). The following may also be employed: 100 c.c. of the milk are boiled with 1.5 c.c. of a 5 per cent. solution of $CaCl_2$, and filtered. A small portion of the filtrate is treated with H_2SO_4 containing 2 per cent. diphenylamin. This mixture is then floated upon concentrated H_2SO_4, when, if nitrates or nitrites be present in the milk, a blue zone will appear at the line of contract of the two liquids. Or, the test may be applied as follows: 1 c.c. of a solution of diphenylamin in H_2SO_4, is placed in a small porcelain dish, and a few drops of the milk allowed to flow down the side into the acid. If the milk contains nitrites or nitrates, a blue color will appear at the line of separation between the acid and the milk. This test is very delicate, and will detect the presence of a very small quantity of impure water. Nitrites and nitrates are not found in milk, even if contained in the food of the cows.

Determination of the Duration of Lactation.—For this purpose Umikoff suggests the color produced by ammonia in human milk. To 5 c.c. of the human milk to be tested, add 2.5 c.c. of a 10 per cent. ammonium hydroxide solution, and warm the mixture to 60° C. for fifteen to twenty minutes. Human milk when so treated assumes a reddish-violet color, the intensity of which increases with the duration of lactation, from rose-violet to dark-brown violet.

Condensed Milk.—Owing to the difficulty of keeping ordinary milk, several processes of preserving it by concentration have been employed. As early as 1837 Newton preserved milk by evaporating it in shallow pans at 50° C. (122° F.), during which time air was blown through the milk. From that time to the present, preserved or condensed milk has been an important article of commerce. When milk is simply evaporated, without the addition of a preservative, it is called **condensed milk.** This is also put into the market sometimes under the name of evaporated cream. This term is also applied to what properly should be termed **preserved milk,** or milk which has been condensed, with the addition of cane-sugar. Preserved milk is much thicker in appearance than condensed. Milk is usually condensed to about one-third its original volume, although the makers usually claim that it is condensed to one-fourth its original volume. Analyses of a large number of samples made at various times in this country give the average as a little short of one-third. The addition of 2 parts of water to 1 of condensed milk should, therefore, produce a milk of the same degree of richness as the whole milk before condensation. Analyses made by Cornwall, of the condensed milks found in the American market, showed the following average :

Water, ...	26.95	per cent.	Milk-sugar, ...	13.38	per cent.
Milk solids, . . .	34.36	"	Cane-sugar, . .	38.82	"
Casein and albumin,	9.25	"	Ash, ...	1.92	"
Fat, ...	9.69	"			

Calculating from these results, he found that the condensation varied from 2.27 to 3.12 times, the average of all analyses being about 2.74 times, or the milk was condensed to not quite one-third the original volume. Condensed milk is largely used as a nourishment for young infants. For this purpose it is usually diluted with about 9 to 12 parts of water. Meigs has shown that if 1 part of the best commercial sweetened condensed milk be mixed with 9 parts of water, the mixture somewhat closely resembles in composition that of human milk, with the exception that it is deficient in fat, and that this mixture, with a small portion of cream added, gives a milk of nearly the chemical composition of human milk. In digestibility, condensed milk is inferior to cows' milk or human milk. It is open to the objections above mentioned to sterilized milk. It is open to the additional objection that a large part of the sugar present, when sweetened milk is used, is cane-sugar instead of lactose, the natural sugar of milk. Cane-sugar more readily undergoes acid fermentation in the stomach or intestine of the infant than lactose. Infants fed exclusively upon condensed milk show a tendency to develop rickets, or, a failure of the nourishment of the bony structures. As a result, the development of the teeth, and the ability to walk are somewhat delayed.

THE URINE.

The urine is an excretory fluid thrown off by animals. It is partially filtered from the blood by the kidneys, and partly elaborated by these organs from waste materials found in the blood. It is composed of a watery solution of certain inorganic salts and nitrogenous principles which are of no further use to the body. As will be seen from the table at the end of this chapter, human urine is not a liquid of uniform composition, but subject to very considerable variations. These variations may be physiological, or they may be indicative of diseased conditions, and a knowledge of them is essential to a correct diagnosis of many diseases.

General Physical Properties.—Normal urine, when fresh, is a clear, amber-colored, transparent liquid, having a peculiar, aromatic, characteristic odor, a bitter, saline taste, a distinctly acid reaction, and a specific gravity of from 1018 to 1022. The average specific gravity is generally given as 1018 to 1020. When it is kept in a clean vessel and away from contact with air, it will undergo but slight changes in several days.

Composition.—The urine is chiefly a solution of urea and certain organic and inorganic salts, holding in suspension epithelial cells and mucus. The composition will be found in the table at the end of this chapter, with the chief variations met with in diseased conditions, and their significance. The urine, like milk and other animal fluids, is not of constant composition. It is influenced by the amount of water and other fluids taken; by the temperature of the skin; by the emotions; by the blood-pressure, local or general; by the amount of work done, the time of day, the age, the sex, the influence of medicine, etc.

Quantity.—The **quantity** of urine passed in twenty-four hours varies considerably. The average daily quantity passed by a healthy adult is from 1400 to 1600 c.c., or about 50 fluidounces. The quantity of total solids contained in this is about 60 gm., or 1000 grs., and about one-half of these solids is composed of urea.

The variations in the quantity will be found in the table at the close of this chapter.

The Color and Transparency.—In health, the color is usually a light amber. In general, the greater the quantity, the lighter the color; and the smaller the quantity, the darker the color. As the color deepens by concentration, it becomes more reddish. The color, as well as the quantity, is subject to great variations, even in health. It may vary from almost as clear as water to a dark yellowish-red, according to the degree of concentration. After drinking large quantities of fluids, the quantity is very much increased and the color is light. After severe sweating, or in abstinence from drinking, it becomes concentrated and darker in color. The normal color of the urine is due to several more or less closely allied pigments, the chief of which are **urobilin** and **uroxanthin.** (See p. 526.) These coloring matters are probably derived from the biliary coloring matters. The abnormal coloring matters are chiefly those of the blood or bile, melanin, hemoglobin, and coloring matters due to medicinal substances, and certain vegetables. An excess of the normal pigments of the urine may be expected in febrile conditions, and in diseases in which the blood-cells are undergoing rapid destruction. Urobilin, when it exists in excessive quantities, colors the urine a dark brownish-red, even without concentration, and the foam of such a urine is of a yellow or yellowish-brown color. There is a marked increase of urobilin in conditions where the hepatic cells fail to perform their proper function; that is, in the condition known as biliousness. In such cases the skin and other tissues may also show the presence of the same yellow color.

The Tests for Urobilin.—First. The spectroscopic examination shows absorption bands in the green, between the lines *b* and *F.* In order to see these lines, it is often best to dilute the urine by pouring water carefully upon the top of the heavier urine in the test-tube. After allowing the liquids to remain at rest for a short time, examine the water above the urine for the absorption bands.

Second. Chemically, we may test for urobilin by the addition of ammonia to the urine, when, if much urobilin be present, it gradually assumes a greenish hue. It is then filtered and a watery solution of $ZnCl_2$ added, when there appears a rose-red color with greenish fluorescence, due to urobilin.

Uroerythrin and **urochrome** have been described as occurring in urine, but little is known of them.

The coloring matters of the bile and the blood will be considered again.

Certain medicines and vegetables, when taken by the mouth, may color the urine. Rhubarb and senna give the urine a brownish color; if made alkaline, it becomes a purple-red. The coloring agent in this case is the chrysophanic acid found in these medicines. After taking logwood, the urine becomes reddish, or violet when made alkaline. Santonin colors it yellow or greenish-yellow, which, on the addition of an alkali, changes to red. Picric acid also gives a yellow color, which does not change to red on the addition of an alkali. Phenol, naphthalin, creosote, preparations of tar, or arsin (AsH_3), impart either a greenish or a greenish-black color to the urine. Salol, resorcinol, antipyrin, and several other coal-tar remedies sometimes cause the urine to assume a violet-red or brown color. Brown or brownish-black urine is observed in patients with melanotic tumors. The coloring matter in this case is melanin.

Transparency.—Normal urine is transparent, containing only a slight flocculent cloud of mucus, visible after standing a few minutes. If the urine is turbid when passed, it is pathological. It is usually turbid in all diseases of the urinary passages, from the excessive amount of mucous and epithelial elements, and because the urine in this condition readily undergoes alkaline fermentation in the bladder, when the earthy phosphates are precipitated as a white sediment. In fevers, the quantity of urine is occasionally so small that the urates separate even in the bladder, and especially is this the case in certain diseases of children, where oxidation is deficient, as in capillary bronchitis and pneumonia. Admixtures of blood, pus, and chyle make the urine turbid. The most striking turbidity is produced by the admixture of chyle, which gives it a milky-white appearance. Here the milky appearance is due to an admixture with the urine of emulsified fat and imperfectly dissolved proteids. Many urines which are clear when passed become turbid on standing, from the separation of the acid urate of sodium or ammonium. The turbidity of alkaline urine has already been mentioned. All urines become turbid on standing for a few days, from the appearance of swarms of bacteria in the solution. Such turbidity can not be separated by filtration through paper. They can be removed by shaking the urine with some insoluble powder and repeated filtration. Powdered glass, silica, or talcum answers the purpose.

Specific Gravity.—This varies from 1015 to 1028, according to the degree of dilution or concentration. Pathological urines may vary from almost that of water to 1050. As a rule, the urine of Bright's disease is of low specific gravity, while in diabetes mellitus, and in all acute fevers, it is of high specific gravity. The specific gravity of urine is generally determined by the urinometer, which is a small hydrometer graduated to include the variations in specific gravity usually found in urine. (See p. 21.) It is usually graduated so that only the last two figures of the specific gravity appear upon the stem, and so as to read correctly at 60° F. If the temperature is above 60° F., it will be sufficiently accurate for clinical purposes to add one degree in specific gravity for every 3° C. (5.4° F.) in temperature—*i. e.*, if it read 1018 at 80° F., it would read 1024 at 60° F. The ordinary urinometers of the market are apt to be unreliable. It is best, therefore, to test the instrument by careful determinations of the specific gravity of solutions of common salt, with the specific gravity flask, and compare the readings of the urinometer with these determinations.

The urinometer is used as follows: The urine is placed in the upright jar, or cylinder, wide enough and deep enough to allow the instrument to float freely. When it has come to rest, the surface of the fluid in the jar is brought to the level of the eye, and the reading taken at the lower edge of the meniscus formed by the upper surface of the urine. The mark on the instrument which is cut by this line, and which can be distinctly seen, is taken as the correct reading. If the urine be turbid, this method can not be employed, as the reading will be more or less uncertain. Should the quantity of urine at hand be not enough to float the urinometer, it may be diluted with an equal volume of water, the specific gravity taken, and the last two figures multiplied by two, to get the true specific gravity.

Reaction.—Normal urine is faintly acid, and grows more acid for a few hours after being voided, due to the so-called "**acid fermentation.**" During this period of acid fermentation there is frequently deposited a whitish or pinkish, or, at times, reddish sediment, due to the separation of the acid urate of sodium or to crystals of uric acid. This sediment disappears again on warming the solution. On standing still longer exposed to the air, the acidity grows less and less, and at the same time an odor of ammonia begins to be developed, and finally the reaction changes from acid to neutral, and from neutral to alkaline, with a strong odor of ammonia and more or less odor of putridity. The rapidity with which these changes take place is dependent upon the composition of the secretion, and upon the temperature, taking place more rapidly in warm than in cold temperatures. An abundance of mucus, which can usually be seen after a few hours as a light, flocculent cloud, settling near the bottom of the vessel containing the fluid, greatly hastens these fermentative changes. This is especially the case if the bladder or the kidneys are in a diseased condition. There is produced with the mucus, especially in diseased conditions of the bladder, a peculiar soluble ferment, which hastens the decomposition of urea and the production of ammonium carbonate.

The reaction of urine is best tested by dropping a small piece of a red and a blue litmus paper into the solution. If both are found red after a few minutes, the reaction is acid. If both are blue, it is alkaline. If they remain unchanged, the reaction is said to be "amphoteric." If the alkalinity be due to ammonium carbonate, the red paper, on drying and warming over a flame, turns red again. If due to the fixed alkalies, it remains blue on drying and warming.

The fermentation of urine is due to certain micro-organisms, of which the **micrococcus ureæ** is the best known. Normal urine is free from these organisms when passed, but in certain abnormal conditions it may undergo an alkaline fermentation while still in the bladder, and that apparently without the intervention of these organisms. It has been found that the fermentation may be complete in the presence of an amount of carbolic acid which is fatal to the development of micro-organisms. It has been assumed that an enzyme is secreted with the thick, mucous secretion of vesical catarrh, which possesses active hydrolytic powers on a solution of urea. As the urine becomes alkaline, from the production of ammonium carbonate from urea, it becomes turbid and acquires a paler color. $CO(NH)_2 + 2H_2O = (NH_4)_2CO_3$. The phosphate of calcium and the ammonium-magnesium phosphate, which separate when the urine becomes alkaline, and to which the turbidity is due, are generally called the **earthy phosphates.** The latter of the two is called the triple phosphate, and is found in nearly all alkaline urines.

Estimation of Acidity.—The acidity of the urine diminishes slightly after a full meal. It follows very nearly the acidity of the stomach contents. It is increased by gastric fermentation and in hyperchlorhydria.

The acidity of the urine increases with the amount of uric acid excreted. The total acidity of normal urine is equivalent to the acidity of 2 to 4 gm. of oxalic acid in twenty-four hours. The greater part of this acidity is due to the acid sodium phosphate produced by the reaction of uric acid upon Na_3PO_4, and the rest is due to the organic acids, most likely lactic.

$$Na_3PO_4 + 2C_5H_2N_4O_3H_2 = 2NaHC_5H_2N_4O_3 + NaH_2PO_4.$$

The acidity may be estimated by a decinormal solution of sodium hydroxide: 25 c.c. of the urine are diluted with 75 c.c. of water in a beaker; 3 drops of an alcoholic solution of phenolphthalein are added, and a decinormal alkali run in from a burette, until a slight pink color is produced. The urine must be fresh for this estimation. Instead of phenolphthalein we may use blue litmus paper as an indicator, taking out a drop of the solution on a glass rod from time to time, and touching the paper. A violet tint indicates neutrality. The acidity may be calculated as phosphoric acid, as acid sodium phosphate, or, as is frequently done, as oxalic acid. Each c.c. of the decinormal alkali is equivalent to 0.0049 gm. of phosphoric acid, or 0.012 gm. (0.01198 more exactly) of acid sodium phosphate, NaH_2PO_4, or 0.0063 of oxalic acid; 100 c.c. of urine should require from 20 to 40 c.c. of $\frac{N}{10}$ NaOH.

Total Solids.—As stated above, the normal amount of solids passed by an adult in twenty-four hours is about 60 gm., or 1000 grs. An approximate estimation of the total solids may be made by multiplying the last two figures of the specific gravity, carefully taken, by the factor 2.33, which will give the number of gms. of solid matter in 1000 c.c. of urine, from which it will be easy to calculate the amount in twenty-four hours. If, for example, the quantity in twenty-four hours be 1500 c.c., and the specific gravity 1020, the total solids would be $20 \times 2.33 = 46.6$ gm. of solids in 1000 c.c. In 1500 c.c. there will be 1.5 times as much, or 69.9 gm. If it be desired to use English measures, we may determine the total solids by multiplying the last two figures of the specific gravity by the number of fluidounces passed. This calculation is based upon the result of numerous exact determinations, which show that the last two figures of the specific gravity very nearly represent the number of grains of solid matter in one fluidounce of urine. Thus, if the number of fluidounces be 50, and the specific gravity 1020, then the total solids will be $50 \times 20 = 1000$ grs. These methods of calculating the total solids give only approximate results, but in most cases will be found sufficiently accurate for clinical purposes.

A more exact method for determining the total solids is to evaporate 10 c.c. in a porcelain dish or watch glass, and dry in a water-oven to constant weight. The difference between the weight of the dish and of the dish with the solids, will give the weight of the solids in 10 c.c. of urine. Even by this method there is some loss during evaporation.

Odor.—The odor of normal urine has been described as aromatic. A putrid odor is due to the products of decomposition. Occasionally the urine is putrid when passed, the putridity being due to the decomposition of pus, albumin, or some other foreign matter mixed with the urine in the bladder. Sulphuretted hydrogen sometimes occurs in the urine, and a fecal odor is occasionally met with, indicating a fistulous opening between the bladder and the intestine, or an abscess between the bladder and rectum. A number of substances, when taken internally, cause the urine to assume a characteristic odor. Many aromatic substances impart their odor, as oil of turpentine (giving the odor of violets), cubebs, copaiba, asparagus, garlic, valerian, etc.

INORGANIC CONSTITUENTS OF THE URINE.

The urine contains certain inorganic salts, especially the chlorides of potassium and sodium, the phosphates of potassium, sodium, magnesium, and calcium, and the sulphates of some of these metals, and several aromatic sulphuric ethers. These salts are generally tested for by the detection of the corresponding acids.

The Chlorides.—For the detection of the chlorides, add a few drops of nitric acid, and then a solution of silver nitrate (1 : 20). The chloride of silver separates as a white, curdy precipitate, which should occupy not more than one-fourth the volume of the urine taken. If the settled precipitate occupies much more or less than one-fourth the volume of the quantity of urine taken, the quantity is increased or diminished. It is always best, in making this test, to compare the specimen under examination with normal urine. In most cases this approximate estimation of the chlorides will be all that the clinician will demand. Occasionally, however, it becomes necessary to make a more accurate determination. For this purpose it is necessary to have a decinormal solution of silver nitrate—*i. e.*, a solution containing 16.96 gm. of pure silver nitrate, dissolved in a liter of distilled water.

Quantitative Estimation of Chlorides.—Dilute 10 c.c. of the urine with about 50 c.c. of water, and add a few drops of a rather strong solution of potassium chromate. Now drop the silver solution from a graduated burette (see Fig. 64) drop by drop, until a permanent reddish color indicates that the chlorine has all been precipitated, and that the silver has begun to form silver chromate. Ten c.c. of urine usually requires 15 to 20 c.c. $AgNO_3$ solution. One c.c. of silver solution represents 0.00354 gm. of chlorine, or 0.00584 gm. of NaCl. In highly colored urines this method is sometimes inapplicable, owing to the change of color being masked by the color of the urine. In such cases it is best to use the following method:

Second Method.—When to a solution of silver nitrate, acidulated with nitric acid, sulphocyanate of ammonium or potassium is added, a white precipitate forms, which is insoluble in nitric acid. If the fluid contains a ferric salt, a blood-red color forms at the moment when the last of the silver is precipitated. **Volhard's** method of estimating the chlorides makes use of this principle. The following solutions are needed in the process: (1) Pure nitric acid. (2) A strong solution of ferric alum (sulphate of iron and ammonia) free from chlorine. (3) A decinormal nitrate of silver solution, made by dissolving 16.96 gm. in a liter of distilled water. (4) A decinormal solution of potassium sulphocyanate, or of ammonium sulphocyanate; this should be of exactly the same strength as the silver solution; it is made by dissolving 6.5 or 7 gm. of the sulphocyanate in about 400 c.c. of water. To standardize the sulphocyanate solution, a portion of it is put into a burette, and 10 c.c. of the decinormal silver nitrate solution brought into a beaker with a few drops of the solution of iron alum. The mixture is well stirred and the sulphocyanate solution added, drop by drop, until a slight but permanent pink color appears. In accordance with the result obtained, the sulphocyanate solution is diluted to such a point that 10 c.c. of it will just neutralize 10 c.c. of the $AgNO_3$ solution. If, for example, 8 c.c. of the sulphocyanate produce a red color, we then know the amount of sulphocyanate in 8 c.c. is that which should be in 10 c.c. Therefore, we dilute the 8 c.c. with a sufficient amount of water to make it 10 c.c., or if we have 450 c.c. we shall add to every 8 c.c., 2 c.c. of water, or we make the calculation by the following proportion:

$8 : 10 :: 450 : x = 562.5$, or 450 c.c. of the solution first made up will require to be diluted to 562.5 c.c., or there must be added 125.5 c.c. of water. Having thus corrected this solution to make it agree in strength with that of the silver solution, we again compare them to see if it is correct.

The process is conducted as follows: 10 c.c. of the urine are measured out with a pipette and placed in a graduated flask of 100 c.c. capacity; 50 c.c. of water are added, and then, successively, 4 c.c. of nitric acid and 25 c.c. of silver solution. The flask is closed with a glass stopper, and agitated until the precipitate ceases to form, and the fluid tends to clear. Distilled water is added, to the 100 c.c. mark. A portion of the fluid is then passed through a dry filter, and to 50 c.c. of this filtrate add a few drops of the iron-alum solution, and then the sulphocyanate solution from the burette until a red color appears. The amount of sulphocyanate solution added, deducted from the amount of silver solution added, gives the amount of silver solution used up by the chlorine in one-half of the 10 c.c. of urine, or 5 c.c.

The calculation is the same as in the first method.

The Phosphates.—Phosphoric acid exists in the urine about two-thirds combined with the alkaline metals and the remainder with lime and magnesium. These phosphates are, therefore, generally distinguished by the terms **alkaline** and **earthy phosphates.**

The **acidity** of the urine is generally believed to be due to the acid sodium phosphate, NaH_2PO_4. Sodium phosphate, Na_2HPO_4. is neutral in reaction, and Na_3PO_4 is alkaline.

In acid urines we have NaH_2PO_4, Na_2HPO_4, $CaHPO_4$, $CaH_4(PO_4)_2$, and $MgHPO_4$; while in alkaline urines we find in solution Na_3PO_4, and as precipitates $Ca_3(PO_4)_2$, $Mg_3(PO_4)_2$, and $MgNH_4PO_4$.

By adding an alkali to normal urine the phosphates of calcium and magnesium, termed earthy phosphates, are precipitated. When NH_4OH is added, all the magnesium present is precipitated as

NH_4MgPO_4. The phosphates of sodium and potassium remain in solution. The earthy phosphates may be approximately estimated by adding a few drops of ammonium hydroxide solution to the urine, and observing the amount of turbidity produced after boiling. This may be quickly done by the use of the centrifuge, in the graduated tube, and measuring the volume of the precipitate. By comparing this with the amount obtained by the same treatment of normal urine, it will indicate whether the quantity is excessive or deficient. The alkaline phosphates may be detected in the filtrate from the earthy phosphates by the addition of a few drops of $MgSO_4$ solution and some NH_4Cl. This precipitate should be about twice as voluminous as that produced by the earthy phosphates, and whether in excess, normal, or deficient, may best be determined by comparison with normal urine. The exact quantitative estimation of the phosphates is rarely required.

Sulphates.—Sulphuric acid occurs in the urine partly in combination with the metals, and partly in combination with certain aromatic bodies of putrefactive origin, called collectively the ethereal sulphates. The most important of these are phenol- and cresol-potassium sulphates, indoxyl- and skatoxyl-potassium sulphates, pyrocatechin- and hydrochinon-potassium sulphates. The two classes of sulphates are generally distinguished as the **preformed** or **mineral sulphates,** and the **conjugate** or **ethereal sulphates.** About nine-tenths of the total sulphuric acid are combined with the metals or are preformed. About one-tenth exists as ethereal sulphates of potassium. (See p. 471.) The preformed H_2SO_4 is detected by the addition of $BaCl_2$ in the presence of free HCl. It appears as a fine, white precipitate of $BaSO_4$, rendering the solution opaque, and milk-like in appearance. An approximate estimate may be made by comparing the turbidity with that of normal urine treated in the same way. An excess of sulphuric acid may be due to the taking of an excessive amount of sulphates with food or drink. It is the ethereal sulphates that have the chief clinical interest. They are derived from the putrefactions of proteids somewhere in the body. In the absence of any other source, an increased quantity of them present in the urine is usually taken as evidence of intestinal putrefaction. They are decomposed, on boiling with dilute mineral acids, into free sulphuric acid and the aromatic substance. The sulphuric acid which they contain is not precipitated in the cold by a slightly acidulated solution of barium chloride. An approximate estimation of the ethereal sulphuric acid may be made by precipitating the urine with an excess of barium chloride and a few drops of hydrochloric acid, filtering off the precipitated $BaSO_4$, and then adding to the filtrate one-tenth its volume of pure HCl, bringing the solution to boiling, and then heating on a water-bath for one hour. The $BaSO_4$ obtained after this treatment of the filtrate represents the sulphuric acid existing as ethereal sulphates.

Potassium Indoxyl-sulphate, or Indican.—$C_8H_6N.SO_4K$. It is easier to test for the aromatic portion of the ethereal sulphates, and make an approximate estimation, than to estimate the sulphuric acid. This is especially true of indoxyl, the most important of them. It is found in normal urine in mere traces. It has a clinical import only when present in increased amount. It then has the same significance as the ethereal sulphates.

Indoxyl may be detected by the addition of an equal volume of strong hydrochloric acid, and dropping into this 2 or 3 drops of a solution of chlorinated soda. Immediately, or after a few seconds, there is formed just beneath the surface a bluish-black cloud of indigo. By stirring the chlorinated soda solution into the urine, we obtain, according to the quantity of indican present, a more or less dark coloration of the whole fluid. If we now shake the fluid with chloroform, the indigo is dissolved out by the chloroform and settles as a blue layer at the bottom. Care must be taken not to add too much of the chlorinated soda. We can judge of the amount of indoxyl by the depth of the blue color.

ORGANIC CONSTITUENTS.

Urea.—$CO(NH_2)_2$. Urea is the most important constituent of the urine, as it is the chief condition in which nitrogen leaves the body. It is by far the most abundant solid ingredient of the urine. Its chemical properties have already been described (p. 455).

Detection.—Urea may be detected by evaporating a few drops of urine on a glass slide, moistening the residue with nitric acid and allowing it to crystallize, and examining the crystals of urea nitrate, $CO(NH_2)_2HNO_3$, under a microscope of low power.

Estimation.—The estimation of urea in urine is a matter of considerable importance, as it is generally looked upon as an index of the nitrogenous metabolism going on in the body, or of the eliminating power of the kidneys. The quantity of urea excreted in twenty-four hours by a healthy adult of 150 pounds body-weight, and doing ordinary work, is usually stated to be from 30 to 33 gm., or from 430 to 550 grs. The quantity will be increased by an increased consumption of nitrogenous food or by hard work, and it will be diminished by a non-nitrogenous diet and by little exercise. In estimating what should be regarded as a normal amount of urea, the condition of the patient, as to exercise, appetite, and dietary, should be taken into account. Roughly, in the absence of sugar, albumin, and other abnormal ingredients, the urea may be regarded as one-half the total solids. The more accurate quantitative estimation requires so little time, apparatus, and skill that it is now very generally employed. The determination is based upon the fact that urea is decomposed by alkaline hypochlorites or hypobromites into carbon dioxide, water, and nitrogen.

$$CO(NH_2)_2 + 3NaBrO = 3NaBr + CO_2 + 2H_2O + N_2.$$

One c.c. of nitrogen at the ordinary temperature and pressure corresponds to about 0.0027 gm. of urea.

The liberated N escapes, and may be collected and measured, while the other products of the reaction remain in solution.

The hypobromite solution is prepared as follows: 100 gm. of NaOH are dissolved in 250 c.c. of water, and to this solution, when cold, 25 c.c. of bromine are added, and the solution kept cold. This solution contains sodium hypobromite, hydroxide, and bromate. The solution should be freshly prepared, as it readily undergoes decomposition. Owing to the instability of this solution and the excessively disagreeable handling of bromine, the author employs a solution of sodium hypochlorite, or chlorinated soda, with the addition of KBr. This solution acts as well, by the method to be described, as the above. Various forms of apparatus have been devised for the quantitative estimation of urea. The simplest of these is the one devised by Dr. C. A. Doremus, which is represented by figure 9. The tube, A, is filled with the above-mentioned solution of hypobromite, and 1 c.c. of urine is introduced with the pipette, B, as nearly as possible at the center of the lower portion of the upright limb. The urea is decomposed and the N rises to the upper, closed end. After the decomposition is complete, the urea is determined by reading the graduations at the surface of the column of liquid. This ureometer, according to the graduation, gives either the milligrams of urea in 1 c.c. of urine, the percentage, or grains per fluidounce.

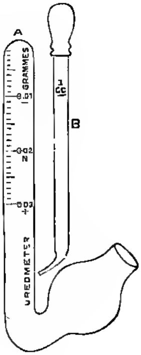

FIG. 9.

The author uses a graduated tube closed at one end. The graduations indicate at once the number of grains of urea in a fluidounce of urine, when 1 c.c. is taken for the estimation. (See Fig. 10.) The ordinary **gas-tube** may be used, when the readings will give the c.c. of nitrogen, from which the urea is calculated.

The process is conducted as follows: A 20 per cent. solution of

KBr is added to the fifth division of the ureometer. Chlorinated soda solution is then added to the fifteenth or twentieth division. The tube is now inclined and pure water poured carefully down the side of the tube and floated upon the top of the fluids already in it; 1 c.c. of urine is then added, in the same inclined position, so that it will not mix with the reagents below, but remain in the water at the surface of the fluid. The tube is now firmly grasped in the right hand, with the thumb tightly pressed upon the open end. The tube is now inverted, and the contents well mixed. A rapid decomposition takes place, which is usually ended in from three to five minutes. During this time the liquid is kept agitated without violent shaking. As soon as the effervescence has ceased, the reading is taken at the surface of the fluid, with the tube still held in the inverted position. It is now opened under water, when the column of fluid in the tube will fall, and the reading is again taken. It is best to have a wide, deep jar for the water, so that the tube may be depressed to bring the surface of the liquid in the tube to the surface of the water in the jar; but an ordinary bowl may be used, as the error caused by the difference in level of three or four inches of water is very slight. The difference in the two readings gives the number of grains of urea in a fluidounce of urine. This quantity, multiplied by the number of fluidounces passed in twenty-four hours, gives the amount of urea excreted in twenty-four hours, which should be not far from 500 grains. A less quantity than 350 grains in an adult of 150 pounds body-weight, who is eating the usual amount, should be regarded as pathological, and suspicious of renal insufficiency, or nephritis.

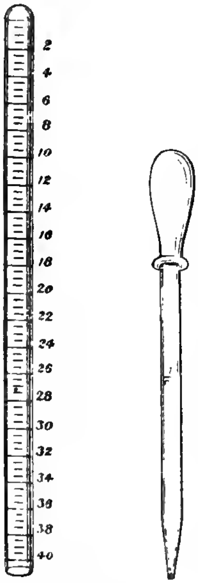

FIG. 10.—UREOMETER.

Uric acid, $C_5H_4N_4O_3$, is a constituent of normal urine, sometimes occurring in the free state, but oftener in combination with potassium, sodium, ammonium, and occasionally with calcium and magnesium, called collectively **urates.** For the description of this acid see page 466.

Uric acid is soluble in 14,000 parts of cold water, and is, therefore, frequently met with as a sediment, and is then detected by microscopical examination. In quantity it varies from 0.4 to 1.0 gm.

(from 6 to 15 grs.) in twenty-four hours. The ratio of uric acid to urea is about 1 : 40 to 1 : 50.

Detection.—It is best recognized, when in the free state, by the microscope. The crystals, as seen with this instrument, are colored yellow or reddish by the pigment uroxanthin, and appear in a variety of shapes, the most common being the "lozenge" or "whetstone" shape. They are sometimes large enough to be seen with the naked eye, when they appear as minute, garnet-colored grains, adhering to the sides or upon the bottom of the vessel.

Chemical Tests.—No. 1, Murexid Test.—Evaporate a portion of the urine to dryness in a porcelain dish upon a water-bath. Moisten the residue with nitric acid, and, after evaporating off the acid, moisten the residue with ammonium hydroxide. If uric acid be present, either in the free state or combined, the residue assumes a beautiful purple-red color, due to the formation of murexid. The reaction is said to occur also with xanthin, hypoxanthin, tyrosin, and some other bodies.

No. 2, Carbonate of Silver Test.—Render the urine decidedly alkaline with Na_2CO_3 or K_2CO_3, and moisten a filter-paper with the liquid. Now touch the moistened paper with a glass rod dipped in a solution of $AgNO_3$. A distinct gray stain indicates the presence of uric acid.

Estimation of Uric Acid.—The estimation of uric acid is usually attended with much difficulty and consumption of time. The author has devised the following volumetric method, which is fairly rapid, reasonably accurate, and requires little skill. It can therefore claim a place as a clinical method. The process is based upon the well-known fact that uric acid is completely precipitated from its solutions containing an excess of ammonium-magnesium mixture and ammonium hydroxide, by silver nitrate. When the precipitation is complete, the slightest trace of silver in solution is shown by the dark color produced in a drop of the clear solution by a soluble sulphide.

The solutions required are:

1. A $\frac{1}{50}$ normal solution of **$AgNO_3$**, made by diluting 1 volume of a $\frac{N}{10}$ solution with 4 volumes of distilled water.

2. **Magnesium mixture**, made to contain about 10 gm. of crystallized $MgSO_4$, 12 gm. of NH_4Cl, and 100 c.c. of aqua ammoniæ (U. S. P.).

3. A solution of **ammonium sulphydrate**, or **potassium sulphide**. This solution should be freshly made, and of such strength that its color is nearly that of the urine.

When the urine contains a sediment of uric acid, or acid urates, it is to be put in solution by warming with a few drops of NaOH solution before beginning the process, and the excess of alkali neutralized with acetic acid. In very dark fever-urines it is best to dilute with an equal volume with water. The titration is performed in a hot solution, to prevent the precipitation of the xanthin bases by silver nitrate.

The process is conducted as follows: To 50 c.c. of the clear urine add 5 c.c. of the magnesium mixture and about 10 c.c. of ammonium hydroxide (sp. gr., 0.960), or enough to give a decided excess. Warm the solution on a water-bath, and add from a burette a $\frac{1}{50}$ normal solution of silver nitrate. From time to time a drop is removed from the solution, by means of a dropper-pipette, with a bit of absorbent cotton wound tightly over the end, so as to make an efficient filter, and after removing the cotton filter, bring a drop of the solution in contact with a drop of the weak potassium sulphide solution on a white porcelain surface. Experiments with pure water showed that it required ½ c.c. of the silver solution in 50 c.c. to give a marked reaction. This amount must therefore be deducted from the reading. The titration is continued until a dark ring or cloud is seen at the line of contact of the two drops, showing the presence of silver in the solution. Each c.c. of silver solution corresponds to 0.00336 gm. of uric acid, and the number of c.c. used (less ½ c.c. for each 50 c.c. of urine), multiplied by this factor, gives the number of grams of uric acid in the urine taken. From this we may easily calculate the amount in 100 c.c. or that excreted in twenty-four hours.

As soon as the process is complete, the precipitate rapidly settles, and it is best to draw off a drop or two from this clear supernatant liquid and test it carefully again. We may also check our work by running in another drop of the silver solution, to see if it produces a cloud, or to see if the precipitation be complete. As there is no excess of silver in the hot liquid at any time, there can be no reduction of silver.

If, after the titration is complete, the solution be cooled, it will usually be found that it will require from 1 to 3 c.c. of the silver solution to again produce the end reaction, because of the precipitation of the xanthin bases as silver compounds. The formula of the xanthin-silver compound is $Ag_2O.C_5H_4N_4O_2$. The factor for the $\frac{N}{50}$ $AgNO_3$ solution is 0.0015—that is, if we calculate them all as xanthin, each c.c. of silver solution used in the cold solution, more than is required by the hot solution, corresponds to the above amount of xanthin bases.

By making two titrations, the one in the hot and the other in the cold urine, we may estimate both the uric acid and the xanthin bases, the latter by the difference in the results of the two titrations.

ABNORMAL CONSTITUENTS OF URINE.

These are albumin, globulin, albumose, peptone, glucose, acetone, diacetic acid, bile-coloring matters, biliary acids, blood, blood-coloring matters, pus, chyle, and abnormal sediments, such as tube-casts, excessive amount of epithelium cells, mucus, etc.

Albumin.—Albumin is found in the urine, at times, without apparent disturbance of health. Usually, however, it is regarded as pathological, and is so often associated with various inflammatory diseases of the kidney that its presence is often taken as evidence of some one of these diseases. It occurs principally in the form of serum-albumin. It is coagulated by a temperature of from 73° to 75° C. (163.4° to 167° F.). In all cases the urine should be clear before applying the tests for albumin. If not clear, it should be either settled and decanted or filtered. It is sometimes necessary to shake the urine with pulverized talc, or other powder, before filtering, to get it clear.* The tests for albumin usually depend upon its coagulation and the formation of a turbidity in the solution. A few tests depend upon a change in color. The tests that are most satisfactory when applied to the urine are as follows:

1. Heat about 5 c.c. of the urine to boiling in a test-tube. It is then examined for even the slightest amount of turbidity. This turbidity, if present, will be due to **albumin** or **earthy phosphates.** Now add, slowly, a few drops of acetic or nitric acid. If the turbidity be due to the phosphates, it disappears; while if due to albumin, it remains permanent. Care must be taken, in the addition of the acid after boiling, to note the effect after each drop is added, and to go on adding until there can be no doubt that the urine is distinctly acid. This test will show traces of albumin under the most favorable conditions.

2. **The Contact Method.**—Place 2 c.c. of pure HNO_3 in a narrow test-tube, and, inclining the tube to one side, pour the urine carefully down the side of the tube so that it may float upon the acid. This is best done with a dropper-pipette, or by pouring the urine from one test-tube into another, holding both in a nearly horizontal position. If this be done carefully, there will be very little admixture of the two liquids. If albumin be present, a white, opaque zone of coagulated albumin appears at the line of contact of the two fluids. A brown zone will frequently be seen at this point, which grows in intensity on standing, and is due to the action of the acid on the coloring matters, but it does not give any turbidity unless albumin is present. If bile be present, the color may be green; if blood, brown-red. This test is decidedly more delicate than No. 1.

Precautions.—Occasionally after the administration of turpentine, or balsams and resins, these are precipitated by HNO_3 as a yellow-white cloud, which, however, is soluble in alcohol. I have never seen the uric acid, sometimes set free by HNO_3, resemble the precipitated albumin nearly enough to be mistaken for it. Roberts modifies this test by using, instead of pure nitric acid, a mixture of 1 volume of HNO_3 and 5 volumes of a saturated solution of $MgSO_4$. This reagent is as sensitive as HNO_3, and pleasanter to handle. It is used in the same way.

3. **Acetic Acid and Potassium Ferrocyanide.**—Acidulate the urine with acetic acid, filter if much mucin is precipitated, and then add a few c.c. of a solution of potassium ferrocyanide. Or, better, float the acidulated urine over the K_4FeCy_6 solution. If albu-

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* Turbid urines may be rendered clear by adding a few drops of $MgSO_4$ solution, then an excess of NH_4OH, and filtering from the phosphates. The urine is then acidified with acetic acid.

min be present, it appears as a white precipitate. This reagent does not precipitate peptone, mucin, or alkaloids. It is a very delicate and reliable test.

4. **Picric Acid with Acetic Acid.**—A cold, saturated solution of picric acid may be used by the **contact method,** after previous acidulation of the urine. At the line of contact the albumin appears as a white zone. Heat afterward to dissolve alkaloids, mucin, peptones, and urates, which are precipitated with the albumin. It is better to heat the urine before adding the test solution.

5. **Sodium Tungstate Solution.**—**Reagent:** Made by mixing equal parts of cold, saturated solutions of sodium tungstate and citric acid. As its specific gravity is heavier than that of urine, it is best applied by the contact method, adding the reagent first. This is an extremely delicate test, and precipitates at the same time peptones, mucin, some alkaloids and urates, all of which, except mucin, are dissolved by heating.

6. **Tanret's Test (Potassio-mercuric Iodide Test).**—Reagent prepared as follows: Mercuric chloride, 1.35 gm.; potassium iodide, 3.32 gm.; acetic acid, 20 c.c.; distilled water, 80 c.c. The $HgCl_2$ and KI are separately dissolved in water and then mixed, and the acetic acid afterward added. The resulting liquid is heavier than urine (sp. gr., 1040), and is best used by the contact method. It is exceedingly delicate, detecting 1 part of albumin in 20,000 parts of urine Heat to dissolve the alkaloids, mucin, and peptone, as in tests 4 and 5.

Jolles suggests the following very delicate reagent: Mercuric chloride, 10; succinic acid, 20; sodium chloride, 10; distilled water, 500.

7. **Acidulated Brine Test.**—**Reagent:** To a pint of a saturated solution of common salt add 1 ounce of HCl, and filter if necessary. This is a delicate test for albumin when properly used. It has a high specific gravity, and is best used as follows: The solution is heated to boiling, and the urine added by the contact method. If albumin be present, it appears as a zone at the contact surface. It does not precipitate peptone, albumose, or the alkaloids.

8. **Trichloracetic Acid.**—This is a white, crystalline acid, sometimes employed as a test for albumin. It may be used in the form of a saturated solution by the contact method, or the crystals may be added directly to the urine, when they will form a strong solution at the bottom of the tube. It presents no decided advantages over the tests above mentioned.

9. A solution of **salicyl-sulphonic acid** in water, or the crystalline acid added directly to the urine, is a very delicate test for all forms of albumin, precipitating albumoses and peptones, but not alkaloids or mucin. The albumoses and peptones dissolve on heating, to

reappear on cooling. A large excess of mucin, or nucleo-albumin, may give a cloudiness with this reagent. It is a white, crystalline acid, formed by saturating strong H_2SO_4 with salicylic acid, and setting aside to crystallize.

10. **Metaphosphoric** or **glacial phosphoric acid** has also been recommended by some as a reliable test for albumin in urine. A solution of **resorcinol** in water (1 : 3) has also been highly recommended.

Albumin test-papers, suggested by Dr. Oliver, may be prepared by saturating bibulous paper in solutions of potassiomercuric iodide, of potassium ferrocyanide, and of citric acid. To use these papers the urine is acidified with one of the citric acid papers, and then either a potassiomercuric iodide or a potassium ferrocyanide paper added.

The **quantitative** estimation of albumin is of considerable importance, but somewhat difficult to perform. Comparative tests are all that the clinician will usually find necessary. The easiest approximate method is to boil a given quantity of the urine in a test-tube, add 2 or 3 drops of nitric acid, set aside for twelve hours, and note the volume occupied by the precipitated albumin. This is generally spoken of as **volume per cent., and has no relation to actual percentage.**

A more accurate method, and one sufficiently so for clinical purposes, is with **Esbach's albuminometer.** This consists of a graduated glass tube, shown in figure 11. To use the instrument, fill to U with urine, and to R with the test liquid. Close the tube by a rubber stopper, mix by agitation, and set aside for twenty-four hours. Each of the main divisions which the precipitate covers represents 1 gm. of albumin in 1 liter of urine.

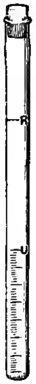

FIG. 11.

Test solution:		
	Picric acid,	10 gm.
	Citric acid,	20 gm.
	Water,	1000 gm.

Comparative tests may be made by adding any of the above reagents to a measured volume of urine, and then placing the tube in the centrifugal machine, separating the coagulated albumin and measuring its volume.

Densimetric Method.—Acidify the urine with acetic acid; take the specific gravity as accurately as possible, noting the temperature. Coagulate the albumin by boiling, and filter from albumin. Bring the filtrate to the same temperature as before, and take the specific gravity again. The difference in specific gravity degrees, multiplied by 0.4, gives the grams in 100 c.c. of urine. Or, a difference of one degree in specific gravity gives 0.400 gm. albumin in 10 c.c. of the urine. It will be seen, therefore, that the specific gravity should be very accurately taken with the picnometer.

Serum-globulin, or **paraglobulin**, is usually associated with serum-albumin, from which it may be separated. It may be detected in the urine as follows: To a large volume of water in a beaker or urine-glass let fall a few drops of albuminous urine. If globulin be present, each drop as it falls will be followed by a milky train, which, when enough is added, forms an opalescent cloud in the water. The addition of acetic acid dissolves this cloud. This test depends upon the fact that globulin is soluble in a weak solution of sodium chloride, such as urine is, but on greatly diluting this solution the globulin becomes insoluble. It is, therefore, precipitated by diluting the urine until the specific gravity is 1002 to 1003.

It may be precipitated by rendering the urine slightly alkaline with NH_4OH, filtering to separate the phosphates, and adding to the filtrate an equal volume of a saturated solution of ammonium sulphate. If a precipitate forms, it is globulin. It occurs with serum-albumin, and rarely without it. It is most abundant in lardaceous kidney, in some cases of acute nephritis, and in the temporary albuminuria of disordered digestion.

Albumoses, or Propeptones.—To test for albumose it is best to first remove the albumin. This is best done by acidifying the urine with a few drops of acetic acid and adding about one-third its volume of a saturated solution of common salt, boiling and filtering. Albumin and globulin are thus removed. The filtrate is allowed to cool, and any turbidity which separates on cooling, or after the further addition of the salt solution, and which disappears by heating to reappear again on cooling, is albumose or propeptone.

Or, after the removal of the albumin and globulin as above, the solution may be saturated with ammonium sulphate, when albumose, if present, will be precipitated. The only disease with which it appears to be associated is osteomalacia.

Peptone.—Peptone is not present in normal urine, but is occasionally found, either with or without albumin. Peptone differs from albumin and albumoses, in that it is not precipitated by tests Nos. 1, 2, 3, 7, and 8, but is precipitated from a cold solution by Nos. 4, 5, 6, and 9, and by tannin, phosphotungstic acid,* and Millon's reagent. When precipitated by tests 4, 5, 6, and 9, it is dissolved when the solution is warmed, and separates again as it cools. Peptone gives with the biuret reaction a rose-red color, while albumin gives a purple or blue.

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* Phosphotungstic acid is made by adding H_3PO_4 to a hot solution of sodium tungstate till decidedly acid. Cool, and render strongly acid with acetic acid or HCl. Filter after standing over night.

Tests for Peptone.—These all require previous treatment of the urine. If albumin be present, it should first be removed, either by saturation with ammonium sulphate and filtration, or by the addition of acetic acid and potassium ferrocyanide, and filtering. It is usually desirable to decolorize the urine by the addition of a solution of lead acetate, as long as it produces a precipitate, and filtering. This treatment is not always necessary. The test may now be applied to this filtrate. Phosphotungstic acid, acidulated with acetic acid, added to this filtrate, or to the solution from which other proteids have been removed, will precipitate peptone, if present; but if it be present in small quantities, the cloudiness appears only after five to ten minutes.

A less sensitive test than the above is made by floating the urine upon some Fehling's test solution. At the point of contact, a delicate rose-red zone will indicate peptone. When positive, this test is valuable; but when negative it will not prove the absence of traces of peptone.

The presence of peptone in the urine, although it is not positively settled, is believed to be due in most cases to the disintegration of pus-corpuscles somewhere in the body, and the absorption of the decomposition products. It is found in many of the acute fevers and in many acute suppurative processes. It may serve to indicate whether a pleuritic effusion is purulent or not, and to distinguish tubercular from epidemic cerebrospinal meningitis, as the latter is usually attended with peptonuria, while the former is not.

Mucin, or Gluco-proteids.—Mucin is secreted by the healthy mucous membranes. It can not be regarded, therefore, as abnormal in the urine until it is present in increased amount, and then it indicates an irritated or inflamed condition of the membranes of the urinary tract. It is not precipitated from its solutions by boiling, but is precipitated by alcohol, dilute mineral acids, acetic, picric, and citric acids. It is best detected, in clear urines, by its forming a sediment on standing, which floats as a translucent cloud near the bottom of the containing vessel, but not upon it. It may also be detected by floating the urine upon a solution of citric or acetic acid, when just above the line of contact a somewhat indefinite zone or coagulum gradually makes its appearance. Albumin, when present, is not precipitated by these acids without the application of heat.

We may also precipitate mucin by the addition of about two parts of alcohol to one part of urine, when mucin and any albuminoid bodies present will precipitate. The precipitate may be filtered out, washed with alcohol, and the mucin dissolved out with warm water, or lime-water, when it may be precipitated from the filtrate again with alcohol or the dilute acids.

Accidental Albuminuria.—Whenever the urine contains blood, pus, or serous discharges it will of necessity contain albumin. **Fibrin** will be found when there are hemorrhages from the genito-urinary passages, and in intense or acute inflammations of the kidneys. It also occurs in the urine of most cases of chyluria. It is readily recognized by its spontaneous coagulation, forming a thick, gelatinous, glairy mass, separating at the bottom of the containing vessel. The coagulum may be filtered out and its solubility determined. If insoluble in dilute alkalies and ten per cent. NaCl solution, it is fibrin.

Blood.—The presence of **blood** may be detected most readily and certainly by the microscope, when the red blood-corpuscles may readily be seen.

Guaiacum Test for Blood.—Mix a small portion of the urine in a test-tube with an equal volume of a mixture of freshly prepared tincture guaiacum and spirits of turpentine. The turpentine should previously have been exposed to the air for some time. If blood-coloring matter be present, the mixture assumes an indigo-blue color, whose rapidity of formation and depth of color depend upon the amount of blood-coloring matter present. Pus frequently, if not always, gives the same color. Saliva and salts of iodine also give a blue color with this test, but the color due to these substances appears only after a considerable lapse of time, and is seldom likely to mislead. From the depth of the color of the urine, and the rapidity of the appearance of the blue color, one can judge of the relative amount of blood present. The spirits of turpentine used in this test may be replaced by a solution of peroxide of hydrogen, or a mixture of ether and H_2O_2 (ozonic ether).

Having determined that blood is present in the urine, it is a difficult matter to decide whether the albuminuria is due entirely to the albumin introduced with the blood, or whether it is a true albuminuria of renal or inflammatory origin. This will often depend upon other symptoms than those to be found in the urine. Dissolved blood-coloring matter is sometimes met with in the urine, when it is called **hemoglobinuria.** In hemoglobinuria, blood-corpuscles are not to be found with the microscope, while in **hematuria** the corpuscles are found. It occasionally happens that the urine rapidly becomes alkaline after being secreted, and the red blood-corpuscles are disintegrated and dissolved by the alkaline urine. The urine containing the dissolved corpuscles is then always alkaline, while the urine of true hemoglobinuria is usually acid. We may conveniently distinguish, then, two conditions: In one, the blood-coloring matter is in solution, and in the other it is in suspension as blood corpuscles. In the former case the coloring matter will not separate on standing, while in the latter there will usually separate, within a few hours, a more or less abundant red sediment. If the hemorrhage be a profuse one, especially if from the bladder or ureters, the blood will almost all of it settle to the bottom of the containing vessel, and leave a clear yellow, almost normal-looking urine above; while if the hemorrhage be a gradual oozing, as in acute inflammation of the kidneys, the coloring matter will remain in suspension and the liquid retain its color for many days. It is not unusual to have hemoglobinuria and hematuria together, especially in acute diffuse nephritis, or in malarial hematuria.

If we add an alkali to urine containing blood, the earthy phosphates are precipitated, carrying down with them the blood-coloring matter and forming a blood-red deposit. By the application of heat the sediment deposits more rapidly, and the solution may assume a green color. If the urine is already alkaline, and the phosphates have separated out, we can produce a precipitate for the purpose of carrying down the blood-coloring matter by the addition of a few drops of a solution of $MgSO_4$. **Hemin crystals** may be prepared from the above precipitates, by spreading a small portion of them upon a glass slide, and treating this with a crystal of common salt and a drop or two of glacial acetic acid, covering with a cover-glass, warming it gently, and examining, after a few hours, with the microscope. The crystals appear as small, oblique plates of a dark-red or brown color. They are easily recognizable by a good ¼-inch lens.

Pus.—If the urine contains **pus** it will always be turbid to the naked eye, and rapidly deposit a white or greenish-white sediment. The clear solution will be found to contain albumin and globulin. The application of heat to the sediment does not dissipate it, as it does the sediment of urates. Neither is it dissolved by dilute acids, as is the somewhat similar-looking precipitate of the earthy phosphates. A whitish sediment, therefore, which is insoluble with heat or dilute acids, and which dissolves in strong alkaline solutions, giving a gelatinous, ropy liquid, is probably pus. (Donné's test.) When pus is treated with a solution of hydrogen peroxide it undergoes rapid effervescence. This is a valuable test for pus in the urine or in other fluids. The microscope is a more certain test for pus. Having detected pus in the urine, it is sometimes very difficult to determine whether the albuminuria accompanying it is accidental—*i. e.*, whether the albumin is derived from the pus, or whether there is a true albuminuria due to some disease of the kidney. The symptoms of the patient will assist in determining in many cases, though not in all.

Sugar.—It has been claimed by many that dextrose occurs in normal urine, and it has been disputed by equally good authority. The most delicate tests do detect glucose in most urines otherwise normal, though not in all. Suffice it to say, that the usual tests, and those here mentioned, except Molisch's, will not detect this substance in normal urine. Its appearance, then, in sufficient quantities to be detected by any of them must be regarded as abnormal.

When glucose occurs in the urine in an appreciable amount, it is known as **glycosuria.** When its occurrence persists for a considerable time and in considerable amount, and is attended with an increased amount of a light-colored urine, generally of a high specific gravity, and an increased daily excretion of urea, it is pathological, and the disease is known as **diabetes mellitus.** The specific gravity is some guide to the detection of diabetes mellitus, but the specific gravity alone is not conclusive. A high specific gravity, with a large quantity of light-colored urine, is strong presumptive evidence of diabetes mellitus. The finding of sugar in such a case is confirmatory. The detection of sugar in the urine is a comparatively simple process.

Tests for Glucose.—Trommer's Test.—To 4 or 5 c.c. of urine, in a test-tube, add one-half its volume of sodium hydroxide solution, and 1 or 2 drops of a solution of $CuSO_4$ (1 to 10). If sugar be present, a clear, deep-blue color is obtained. If an excess of copper sulphate be added, a clear solution may not be obtained, and will, in this way, disturb the test. The solution is now to be heated almost to boiling, but it is better not to boil. If sugar be present, at first a greenish and then a yellowish turbidity forms, which rapidly changes to a reddish-yellow color, and precipitates red cuprous oxide. A flocculent precipitate of the earthy phosphates always forms on adding the alkali, and must not be mistaken for suboxide of copper. Urine containing uric acid, the xanthin bases, creatinin, indoxyl-sulphates, peptone, lactose, glycuronic and glycosuric acids, mucus, and other substances found in some urines will decolorize the blue solution, but there will be no red precipitate. In fever urines, this decolorization without precipitation interferes greatly with the employment of this test. It is, therefore, not to be relied upon in doubtful cases.

To eliminate this source of trouble with the copper test, it is best to use a solution of the acetate of copper, or sodium acetate added to the sulphate, to precipitate the uric acid, xanthin, hypoxanthin, and the most of the creatinin and phosphates, filter, and apply the test to the filtrate.

From 7 to 8 c.c. of the urine are heated to boiling in a test-tube, and, without filtering from any precipitate that may form, adding 1 c.c. of the copper sulphate solution; then, when partially cooled, 1 to 2 c.c. of a saturated solution of sodium acetate, having a slight acid reaction, and filter. To the filtrate, which should have a greenish-blue color, add the alkali, or, better, the alkaline tartrate solution used to make Fehling's solution, and boil for fifteen to twenty seconds. Thus modified, the copper test is much more reliable. Most of the interfering substances may be separated by adding to the hot urine one-fourth its volume of a 10 per cent. solution of lead acetate, filtering off the precipitate, and testing the filtrate for sugar.

2. **Other Forms of the Copper Test.—Haines' solution** is made by dissolving copper sulphate in a mixture of equal quantities of glycerin and water. This solution may be used in larger quantities than the aqueous solution of copper used in Trommer's test, and some of the difficulties of that test overcome. The decolorizing effect of normal urine is not sufficient to decolorize a large amount of copper solution. By adding a considerable amount of Haines' solution before heating, this error is partially eliminated. **Fehling's solution** is sometimes employed as a qualitative test, but usually only as a quantitative test; Haines' solution has all the advantages of Fehling's, with the additional advantage that it keeps well.

3. **Bismuth Test.**—To a few c.c. of the urine, in a test-tube, add an equal volume of sodium hydroxide, and then a fragment of bismuth subnitrate; mix well and boil for from three to five minutes. If sugar be present, black metallic bismuth will be deposited as a sediment. If the quantity of sugar be small, only a part of the bismuth will be reduced, and the precipitate will appear gray. Albumin must be removed before this test is applied, or it will be decomposed by boiling with the alkali, forming the sulphide of bismuth, which will give a black precipitate.

4. A better form of this test is as follows: A solution is made of bismuth subnitrate, 2 gm.; Rochelle salt, 4 gm.; sodium hydroxide, 8 gm.; and distilled water, 100 c.c. The urine is heated to boiling and a few drops of this alkaline solution of bismuth added, and, on continuing the boiling, if sugar be present, the mixture turns black. As in the previous test, albumin must be absent before this test is applied. This reagent is exceedingly delicate, and it is claimed to detect 0.025 per cent. of glucose.

5. **Picric Acid Test.**—To about 5 c.c. of urine add one-half as much of picric acid solution (as in testing for albumin), and then 2 c.c. of sodium hydroxide, and boil. If sugar be present, a dark, mahogany-red color is developed. If no sugar is present, a dark hue is developed before boiling, but not the dark color above described. If albumin be present, a turbid ity will form on the addition of the picric acid, but it does not interfere with the test.

6. **Moore's Test.**—Add to the suspected urine one-half its volume of sodium hydroxide solution, and boil. If sugar is present, a dark-yellow, brown, or chocolate color is produced. The depth of color is proportional to the amount of sugar present.

7. **Indigo-carmine Test.—Reagent**: Mix 1 part of indigo-carmine, or of dried commercial extract of indigo, with 30 parts of pure, dry sodium carbonate. To 5 c.c. of the suspected urine add enough of the above powder to give a transparent, blue solution, and heat to boiling *without agitation*. If sugar is present, the solution changes to violet, cherry-red, and finally yellow. On agitation, these colors appear in the reversed order.

Instead of extract of indigo, a solution of sulphate of indigo with an excess of sodium carbonate may be employed. None of the ordinary constituents of the urine affect this test, while many substances occurring in the urine affect Fehling's solution. Many other substances which reduce the alkaline copper solution do not affect the indigo-carmine test. In careful hands it is to be recommended as a sensitive and reliable test for glucose in the urine.

8. A solution of **methylene-blue** (0.333 gm. per liter) has been used as a test for sugar. Five c.c. of this solution are mixed with 2 c.c. of sodium hydroxide, 2 c.c. of urine added, and the solution is boiled for one minute. If sugar be present, the blue color is discharged, but returns on standing.

9. **Safranin** is another coloring matter used as a test for sugar. Mix equal volumes of sodium hydroxide, safranin solution (1 gm. to the liter of water), and the urine, and heat to boiling. If sugar be present, the red color is changed to a yellow. Uric acid, creatin, creatinin, chloral, and chloroform do not give the test. Albumin must be removed. The test seems reliable.

10. **Phenyl-hydrazin Test.**—For the details of this test see **glucose**, in part v of this book.

11. **Alpha-naphthol Test.—Molisch's Test.**—To 1 c.c. of the urine add 2 c.c. of a 10 per cent. solution of α-naphthol in pure methyl- or amyl-alcohol, and after mixing add an excess of H_2SO_4. If sugar be present, a deep-violet color is developed. On dilution with water a blue precipitate occurs, which is soluble in alcohol, ether, and potassium hydroxide, to give a yellow solution. If, instead of naphthol, we use thymol or menthol, a deep-red color is produced, which gives a carmine-red, flocculent precipitate on dilution, which dissolves as above with the formation of a yellow solution. This test is exceedingly delicate, and reacts with most sugars and glucosides. Urea, indican, creatinin, xanthin, uric acid, hippuric acid, phenol, and pyrocatechin give negative results. As many normal urines respond to this test, Molisch concludes that they contain sugar.

The Quantitative Estimation of Dextrose.—This is generally made with Fehling's solution. This solution is best prepared in two parts, which are kept separately, as the completed solution does not keep well. These solutions are prepared as follows: No. 1. 34.639 gm. of pure, recrystallized copper sulphate are dissolved in distilled water, and made up to exactly 500 c.c. No. 2. 175 gm. of crystallized Rochelle salt and 60 gm. of sodium hydroxide are dissolved in distilled water and made up to exactly 500 c.c. When needed for use, exactly equal volumes of these two solutions are mixed. The solution will be of such strength that 10 c.c. are decolorized by 0.050 gm. of dextrose or diabetic sugar.

The process is conducted as follows: Ten c.c. of Fehling's solution are measured out into a beaker or porcelain basin, diluted with about 40 c.c. of water, and brought to the boiling point. The urine is delivered into this blue solution from a burette, until the blue color is just discharged. The amount of urine added is then read off from the burette, and this amount contains 0.050 gm. of sugar. From this it is easy to calculate the quantity contained in 1000 c.c., or a liter. If the urine contains a considerable quantity of sugar, it will be necessary to dilute it with four volumes of water before beginning the titration, when the results of the titration should be multiplied by five. It is always somewhat difficult to determine the exact disappearance of the blue color, owing to the presence in the solution of the precipitated suboxide of copper. This difficulty may be overcome by the addition of some substance that will prevent the precipitation of the cuprous oxide, as NH_4OH, KCy, or $K_4Fe(CN)_6$.

The **author's method** is as follows: Ten c.c. of Fehling's solution are measured out into a suitable flask. To this 10 c.c. of a freshly prepared 10 per cent. solution of potassium ferrocyanide are added, and about 30 c.c. of water. The mixture is heated on a water-bath, and the urine, previously diluted with water, if it contains much sugar, is run in from a burette, drop by drop, until the blue color just disappears. This can readily be seen, as the solution remains clear to the end of the reaction. The addition of the slightest excess of sugar shows itself by the solution becoming quickly brown. By careful comparative tests the author has found this method to be accurate, reliable, and rapid, provided the solution be not boiled during the reduction. The best temperature for the process was found to be between 80° and 90° C. (176° to 194° F.).

Estimation by the Polariscope.—This is a convenient and rapid method for the determination of glucose, when the quantity exceeds 1 per cent., and when all the appliances are at hand, which is seldom the case except in well-equipped laboratories. The method, briefly, is as follows: The suspected urine, freed from albumin, is treated with a solution of basic lead acetate, in the proportion of 1 to 10 of the urine, and filtered. The observation-tube of the polariscope is filled with this fluid, when it is placed in position, and the rotation determined.

The readings must be increased by one-tenth (allowance for the lead acetate solution).

The specific rotatory power of dextrose is + 52.5°. (See p. 52.)

The weight of the sugar in the solution will be given by the formula: $W = \frac{a}{52.5 \times l}$; in which a = observed rotation, l the length of the tube in decimeters, and W the weight of sugar in 1 c.c. of the solution.

Suppose, in a given case, the rotation observed was 4°, after allowing for the lead solution, and the length of the observation tube was two decimeters. We then have $W = \frac{4}{52.5 \times 2}$, or $\frac{2}{52.5} = 0.038$ gm. in 1 c.c. of urine, or 3.8 per cent.

As levulose sometimes occurs with dextrose in cases of diabetes, and as it will rotate the plane of polarized light to the left instead of to the right, and, in fact, as there are a number of substances likely to occur in the urine which rotate the plane of polarized light, this method of determining sugar is not free from error.

Lactose, or milk-sugar, occurs in the urine of nursing women or of women soon after weaning. Its recognition requires first its separation from the fluid.

Dextrin has been found in the urine of diabetics, where it seems to take the place of dextrose. Other carbohydrates found rarely in the urine are pentoses, maltose, and animal gum.

Acetonuria.—Normal urine contains traces of acetone, but it occurs in excessive quantities as a pathological condition. It is found in many of the fevers, certain forms of cancer, in starvation, and in certain nervous troubles accompanying diabetes. The commonest of these is febrile acetonuria. The appearance of acetone in diabetes indicates an advanced stage of the disease, but it decreases in diabetic coma. It is always associated with an increased proteid metabolism, and is looked upon as a product of proteid decomposition with deficient oxidation. It is closely allied to certain other substances found in some urines. These are hydroxybutyric acid, aceto-acetic acid, also called diacetic acid. The following formulæ will show the relations of these bodies:

Hydroxybutyric acid, ...	$CH_3—CH(OH)—CH_2—COOH$.
Aceto-acetic acid (diacetic acid), . .	$CH_3—CO—CH_2—COOH$.
Acetone (dimethyl-ketone), ...	$CH_3—CO—CH_3$.
Aldehyde, ...	$CH_3—COH$.
Acetic acid, ...	$CH_3—COOH$.

Detection.—**Legal's Test.**—Four or five c c. of the urine are treated with a few drops of a freshly made solution of sodium nitroprusside, and then with a strong solution of NH_4OH. The red color produced, which appears in from five to ten minutes of acetone be present, gives place to a purple or violet color on the addition of acetic acid. For a more accurate test it is necessary to distil the urine, and apply this or the following test to the distillate:

Lieben's Test.—To several cubic centimeters of the distillate add a few drops of a solution of iodine in potassium iodide, and then a solution of KOH. If merely a trace of acetone be present, a precipitate of iodoform crystals is deposited. This test is reliable and delicate in the absence of lactic acid and alcohol, but if NH_4OH be used for the KOH, alcohol will not form iodoform, while the acetone will do so.

Diacetic acid appears in the urine of diabetics and of certain fevers, and is always an abnormal constituent. It is most common in the contagious fevers of childhood, and in such cases has little significance; but in adults it is a grave symptom, as it usually precedes the advent of coma. It usually occurs together with acetone, and in the presence of ferric chloride produces a wine-red color, which is not entirely characteristic, because other substances produce the same color. The following process will serve for its detection: A fairly strong solution of ferric chloride is cautiously added to the urine, and if a phosphate precipitates, this is removed by filtration, and more Fe_2Cl_6 added to the filtrate. If a red color appears, it is divided into two portions. One portion is boiled, whilst the other is treated with H_2SO_4, and extracted by shaking with ether. Urine that has been boiled loses its red color, this color being destroyed by boiling. After treatment with H_2SO_4 and shaking with ether, the aceto-acetic acid will be found in the ether. The ether may now be evaporated, and the residue treated with Fe_2Cl_6 solution. when the violet-red color will be obtained if it be present. The urine for this test must be fresh, as diacetic acid is rapidly converted into acetone on standing. Acetone is an oxidation product of diacetic acid. If the quantity of acetone be large, it may cause toxic symptoms.

Hydroxybutyric acid is found in the blood of diabetic patients, and its oxidation produces diacetic acid. The relation of these three bodies is then oxybutyric acid, diacetic acid, and acetone, in the order named. It gives the same color-reaction with Fe_2Cl_6 as diacetic acid. It is best to separate it from the urine with ether, as above described for diacetic acid, before applying the test.

Lipaciduria is a term which has been applied to the condition in which volatile fatty acids are found in the urine. These occur normally in traces. especially formic, acetic, and butyric acids. As a symptom of disease, however, they are of no significance. Formic, acetic, propionic, and butyric acids have occasionally been detected in the urine of fevers, in certain diseases of the liver, and in diabetes. For their detection the urine is distilled with phosphoric acid, and the test applied to the distillate. For simpler tests we may apply the following: Acetic acid may be detected by the odor of acetic ether when the distillate is warmed with alcohol and sulphuric acid. Ferric chloride gives a red tint, which disappears on boiling if acetic acid is present. Formic acid gives a white precipitate with silver nitrate, which blackens on warming.

Fat.—Fat occasionally occurs in the urine, and gives to it a more or less turbid appearance, which clears on shaking the solution with ether. On separating and evaporating the ether, the fat remains behind. In chyluria the opacity is due both to the fat and to albuminous substances in imperfect solution. In some cases the appearance of this turbidity is intermittent, appearing only at certain times of the day; in others it is constant. In some cases chylous urine deposits a spontaneous clot of fibrin, while in others it does not. The fat may be separated by extraction with ether, but the turbidity still remains.

In some rare cases, however, the turbidity disappears with the extraction of the fat.

Detection.—Its detection is usually sufficiently easy from the milky-white color, and the separation of the fat on standing. Microscopically, the fat globules can be detected in some cases, but in others the microscope fails to reveal them. The author has seen a case where they were not visible with a $\frac{1}{16}$-inch objective.

Bile.—Urine containing bile usually has an abnormal color—either a brilliant yellow, a greenish-yellow, or brown. When the bile is abundantly present, the froth or foam produced on shaking the urine is quite permanent, and is more or less colored. A piece of filter-paper or linen moistened with such urine retains the yellow color on drying.

Gmelin's Test.—Upon 1 or 2 c.c. of a partially decomposed yellow nitric acid, in a test-tube, carefully float 4 or 5 c.c. of the suspected urine. If bile-coloring matters be present, a succession of colors will appear in the urine, beginning with green, then passing through blue, violet, red, and yellow, the green appearing at the top and the others below, in the order named. The green color is always present when bile is present, but the reddish-violet color must not be taken for evidence of bile, as the normal coloring matters of the urine may produce such a coloration.

If the decomposed nitric acid, or nitrous acid, be not at hand, it may readily be prepared by adding a fragment of zinc to ordinary pure nitric acid. This test may also be applied as follows: The urine may be mixed with a concentrated solution of sodium nitrate, and the mixture floated upon sulphuric acid, when the play of colors will be obtained as before; or a crystal of sodium nitrate may be dropped into strong sulphuric acid, and the urine floated upon this.

Tincture of Iodine Test.—Upon the surface of the urine in a test-tube, float a few drops of tincture of iodine. At the line of contact of the two fluids there appears, after a few minutes, a beautiful emerald-green zone when biliary coloring matters are present. This test seems to be delicate and reliable.*

Biliary Acids.—While the acids usually occur in the urine of jaundiced patients, along with the coloring matters, their detection is not so easy. We may use the following test: Evaporate the liquid to dryness, and treat the residue, consisting of biliary salts, with alcohol, and filter. After evaporating the alcohol, apply Pettenkofer's test (see p. 468) to a solution of the residue in water. **Dr. Oliver's**

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* Jolles says that antipyrin in urine gives a similar green color.

peptone test is, however, applicable to urine. The reagent is prepared as follows: Pulverized peptone, 2 gm.; salicylic acid, 0.250 gm.; acetic acid, 2 c.c.; distilled water, sufficient to make 250 c.c. The urine, rendered perfectly clear by filtration, is rendered acid and diluted until the specific gravity is 1008. One c.c. of this diluted urine is run into about 4 c.c. of the above test solution. If biliary salts are present, a distinct milkiness promptly appears, but it becomes more intense in five minutes. Albumin, if present, should be separated before the application of this test. The test is very delicate, and apparently reliable.

Diazo-reaction.—This reaction is one that is obtained in the urine of persons suffering from certain specific fevers, especially typhoid fever, measles, septicemia, and in some cases of phthisis. The reagent is made as follows: One gm. of sulphanilic acid is dissolved in a mixture of 350 c.c. of water and 15 c.c. of hydrochloric acid. A second solution is made by dissolving 0.5 gm. of sodium nitrite in 100 c.c. of water. Five c.c. of urine are mixed with an equal volume of sulphanilic acid solution, and then with 3 or 4 drops of the sodium nitrite solution, and, finally, 10 drops of ammonia water. Normal urine shows with this test a yellow or orange color, and a precipitation of phosphates. In certain of the above-named diseases, especially in typhoid, the urine gradually assumes a carmine-red color. The froth produced on agitation is also distinctly red, and the precipitated phosphates show a green or violet color. Many phenol derivatives give a similar color-reaction with the above test, and may lead to erroneous conclusions. According to Ehrlich, this reaction is characteristic of the urine in typhoid, measles, and acute tuberculosis. Others deny the value of the test, the difference being possibly due to the interference of phenol derivatives.

Ferments Found in the Urine.—Pepsin, trypsin, and a diastasic ferment have been found in the urine, in addition to the organized ferments of lactic, butyric, and acetic acids, and urea. The pepsin ferment of the urine is said to be absent in the urine of typhoid fever, carcinoma of the stomach, and, according to some, in nephritis.

Detection.—Pepsin is best detected by Sahli's method. A little pure fibrin is placed in the urine and set aside for several hours. It is then removed, placed in diluted HCl (0.2 per cent.), and the mixture kept at a temperature of from 30° to 40° C. (86° to 104° F.). Any pepsin present in the urine is taken up by the fibrin, and the latter is slowly digested in the acid fluid.

The diastasic ferment is detected in the usual manner by its effect upon starch-mucilage. The milk-curdling ferment has occasionally been found in the urine. (See also Organized Ferments, p. 557.)

Ptomaines, or diamins, have been found in healthy urines as well as in morbid urines. In most fevers, especially in the specific and contagious fevers, the urine contains certain poisonous alkaloids. These can be detected by first acidifying the urine and filtering from any mucus present, and then precipitating with the double iodide of potassium and mercury. The precipitate, which contains these bases, is distinguished from albumin or other substances by its solubility in alcohol at a gentle heat

The **diamins** of the urine may be precipitated as benzoyl compounds by benzoyl chloride and caustic potash. By this means cadaverin, putrescin, and other diamins have been detected in the urine of vesical catarrh. Normal urine is said to be free from these bodies.

URINARY DEPOSITS OR SEDIMENTS.

Normal urine is clear, but on standing it will usually deposit more or less sediment. Urine that is turbid when passed will usually deposit a sediment, which may contain mucus, pus, blood, chyle, earthy phosphates, acid urate of sodium, or an abundance of epithelial cells from the kidney, ureters, or bladder. A turbidity which appears within a few hours after the urine is voided is most likely to be due to acid urates, the oxalate of lime, or the earthy phosphates. When

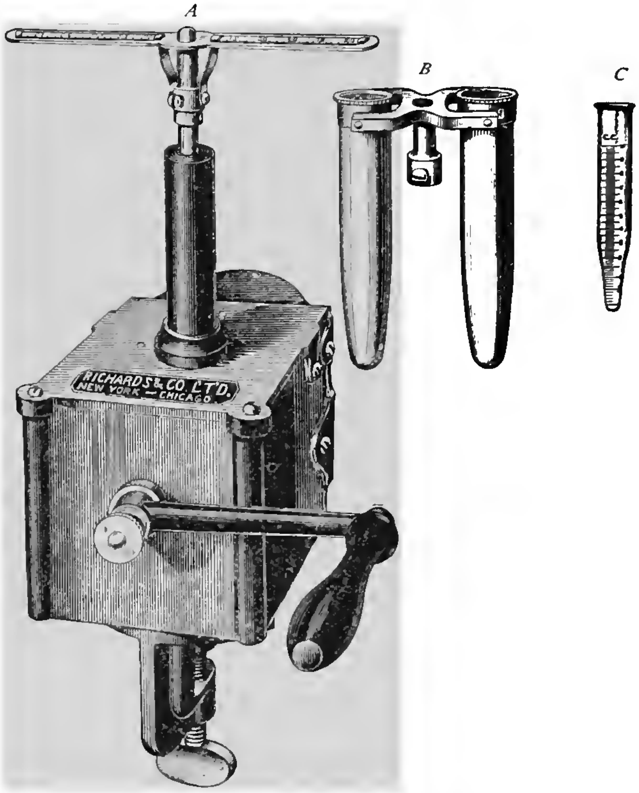


FIG. 12.

such a deposit is to be examined, a few ounces of the urine are set aside in a cylinder or tall vessel to allow the sediment to accumulate, or, better, the sediment may be separated from the fresh urine by the centrifugal machine. The urine to be examined is well shaken and poured into specially constructed tubes (*C*, figure 12); these are placed in the receptacles (*A*) and revolved. The sediment forms at the bottom of the tube. This is more satisfactory than spontaneous sedimentation, because some sediments undergo changes on standing in contact with urine. The usual form of centrifuge is shown in figure 12. It requires from three to four minutes to obtain a complete sedimentation of ordinary urine. The sediment may be removed from the solution by means of a pipette, or narrow glass tube, by holding the finger upon the upper end until it is depressed to the bottom of the glass, and then, on removing the finger for an instant, the sediment will be drawn up into the tube, when it may be removed for examination. It is more convenient, for the microscopical examination of urinary sediments, to **dispense with the usual cover-glass**, as a larger field is available for search for characteristic objects. To the **crystalline deposits** belong uric acid, urates, calcium oxalate, the phosphates or carbonates of magnesium and calcium, cystin, hippuric acid, leucin, tyrosin, etc. **Organized deposits** include mucous corpuscles, blood, pus, casts, epithelium, fungi, and bacteria.

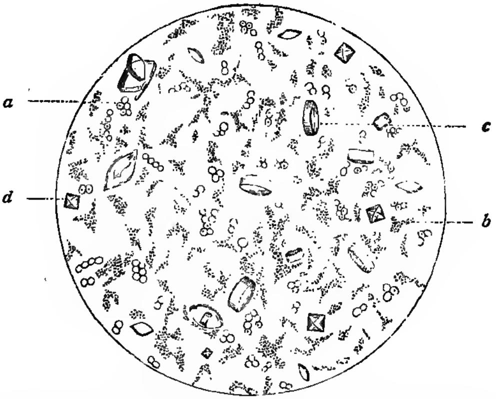


FIG. 13.—DEPOSIT IN "ACID FERMENTATION" OF URINE. (*a*) FUNGUS; (*b*) AMORPHOUS SODIUM URATE; (*c*) URIC ACID; (*d*) CALCIUM OXALATE.

The chemical examination of the deposits should be preceded by a microscopical examination. In fact, with a little experience, the microscopical examination may greatly assist in the chemical examination. Most of the unorganized and crystalline sediments may be easily recognized by microscopical better than by chemical means.

Crystalline Deposits.—**Uric acid** occurs in crystals, differing much in form and size, and stained a brownish-yellow to a light lemon-yellow color by uroxanthin. They are sometimes large, and when grouped together, as at *d*, figure 14, are large enough to be seen with the naked eye. They dissolve when warmed with NaOH solution. The most characteristic forms are those shown in figures 13 and 14.

Acid Urates.—Amorphous urates consist principally of acid sodium urate. (See Fig. 13.) The deposit is amorphous unless a very high magnifying power is employed. Then it is seen to be made up of minute globular particles of yellow, red, or brown color. This sediment separates only from acid urines. It dissolves to a clear solution on adding a solution of NaOH or KOH, or when heated.

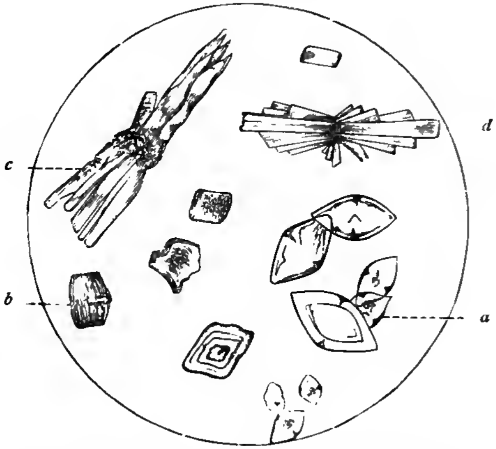


FIG. 14.—URIC ACID. (*a*) RHOMBIC TABLES (WHETSTONE FORM); (*b*) BARREL FORM; (*c*) SHEAVES; (*d*) ROSETTES OF WHETSTONE CRYSTALS.

For the purpose of testing the solubility of the sediment under the microscope, it will be found convenient to place a drop or two of the solvent on the slide at one side of the cover-glass, and put on the other side a piece of bibulous paper. In this way the fluid is drawn under the cover-glass on the one side and removed at the other, the old liquid being replaced by the new. In this way the action of the reagent upon urinary sediments may be readily observed.

Acid sodium urate sometimes crystallizes during the acid fermentation, in the form of larger spheres made up of elongated crystals. They appear under the microscope as yellow or brown, frequently almost opaque spheres, with one or more spicules. When the urine becomes alkaline from fermentation, the amorphous urates are gradually converted into ammonium urate, which has the appearance seen in figure 15.

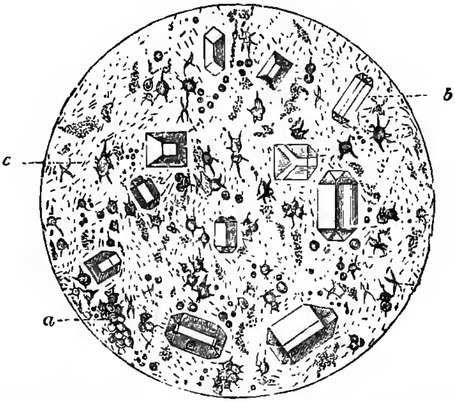


FIG. 15.—DEPOSIT OF AMMONIACAL URINE (ALKALINE FERMENTATION). (*a*) ACID AMMONIUM URATE; (*b*) AMMONIO-MAGNESIUM PHOSPHATE; (*c*) BACTERIA.

Calcium oxalate occurs as a sediment in transparent, strongly refracting, regular octahedrons, which are readily soluble in HCl, but insoluble in acetic acid. They frequently accompany uric acid crystals, and deposit during the acid fermentation, as shown in figure 13.

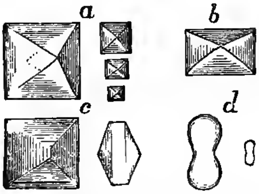


FIG. 16.—OXALATE OF LIME. (*a*) OCTAHEDRA; (*b*) BASAL PLANE OF AN OCTAHEDRON FORMING A RECTANGLE; (*c*) COMPOUND FORMS; (*d*) DUMB-BELLS.

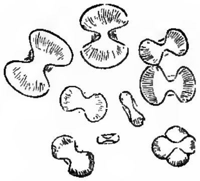


FIG. 17.—PERFECT DUMB-BELL CRYSTALS OF OXALATE OF LIME.

They are frequently called envelope-shaped crystals, from the fancied resemblance to the reverse side of an envelope. They are usually of very small size, and occasionally appear in the form of dumb-bells. (Figs. 16 and 17.) A few isolated crystals of calcium oxalate have no clinical significance. They greatly increase after eating such vegetables as tomatoes, fresh beans, beet-root, asparagus, apples, grapes, honey, and after the administration of rhubarb, senna, squills, etc. Another source of oxalic acid in the body is incomplete oxidation of carbohydrates and proteid, retrograde, decomposition products. For this reason it is frequently met with in excess in diabetes mellitus. It is frequently excessive in fermentative disturbances in the intestinal canal, and in certain nervous disturbances. The long-continued excretion of an excess of oxalate of calcium frequently irritates the kidneys, producing albuminuria, and grave nervous disturbance, and may lead to the formation of calculi, especially renal calculi. It is usually associated with an excessive amount of uric acid, mucus, and phosphates.

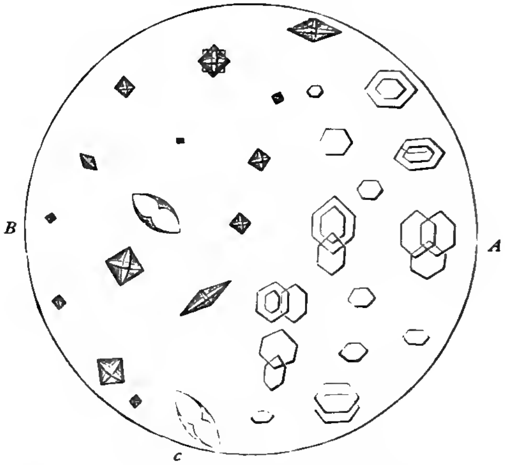


Fig. 18.—*A*, Crystals of Cystin; *B*, Oxalate of Lime; (*c*) Hour-glass Forms of *B*.

Ammonium-magnesium phosphate (triple phosphate) occurs as a sediment in neutral or in alkaline urine. The crystals are large, transparent, highly refracting prisms, usually in the form seen in figure 20. Occasionally it occurs in the form of feathery crystals, or star-shaped groups. They are never colored. They frequently attain a size sufficient to render them visible to the naked eye, especially in a strong light.

Magnesium phosphate is occasionally deposited in concentrated urines of feebly alkaline reaction.

Calcium phosphate crystals appear as pointed, wedge-shaped prisms, either singly or in clusters. They are dissolved by acetic or hydrochloric acid.

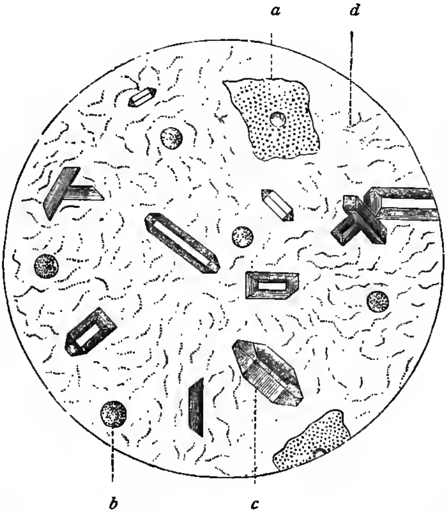


Fig. 19.—Deposit from a Case of Inflamed Bladder (Ammoniacal Fermentation). (*a*) Detached Epithelium; (*b*) Pus-corpuscles; (*c*) Triple Phosphate; (*d*) Micrococcus Ureæ.

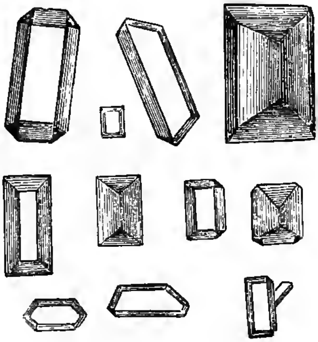


Fig. 20.—The More Usual Forms of Triple Phosphate. × 300.

Calcium sulphate is rarely present as a urinary sediment. It occurs in the form of long, colorless needles or prisms, or in elongated tables with abrupt extremities.

Calcium carbonate occasionally occurs in the urine as an amorphous deposit, but on higher magnification it is seen to be made up of minute spherical granules. (See Fig. 23.)

Hippuric acid occasionally occurs as a sediment in the urine in the form of four-sided prisms, either occurring separately or in groups. They are soluble in ammonia, insoluble in HCl. It occurs especially after the administration of benzoic acid and after eating certain fruits, as cranberries, bilberries, etc. It is of no diagnostic importance.

Cystin.—The crystals of this body appear as regular hexagonal plates, superimposed or contiguous to one another. (See Fig. 18.)

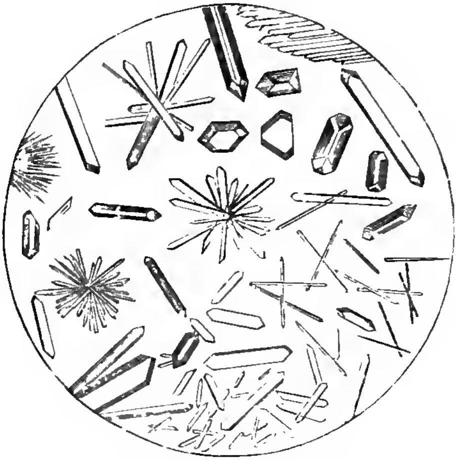


FIG. 21.—HIPPURIC ACID.

They are insoluble in acetic acid, but soluble in ammonia. It is sometimes also found in solution in the urine. Cystin is a decomposition product of proteid matter, and generally the result of bacterial action in the intestines. It is frequently associated with diamins and ethereal sulphates. It sometimes forms calculi.

Leucin and **tyrosin** always occur together. Tyrosin occurs in the sediment in the form of sheaves of very fine crystals. **Leucin,** commonly associated with tyrosin, is more soluble, but occurs to some extent in the sediment in the form of small spheres, not unlike oil globules, which in a good light are seen to be marked with radiating striæ. When quite pure, leucin crystallizes in delicate plates, but as a urinary sediment it usually forms spheres. (See Fig. 22.) Tyrosin has been found in the urine, together with leucin, in phosphorus poisoning, in acute yellow atrophy of the liver, in leukemia, and in some of the infectious diseases.

Fat is deposited in the form of strongly refracting globules of varying size, and readily soluble in ether. It may be present in the urine in small quantities after the fracture of bones, and in some chronic cases of Bright's disease attended with fatty degeneration. In chyluria it occurs in greater abundance.

Indigo occasionally occurs as a sediment in concretions and amorphous fragments, and also in the form of blue crystals and clusters of fine, blue needles. The crystals of indigo are not rare in decomposing and fermenting urines, in which they result from the decomposition of the indoxyl-sulphate. They occur more especially in the urine of hepatic abscess and in cirrhosis of the liver.

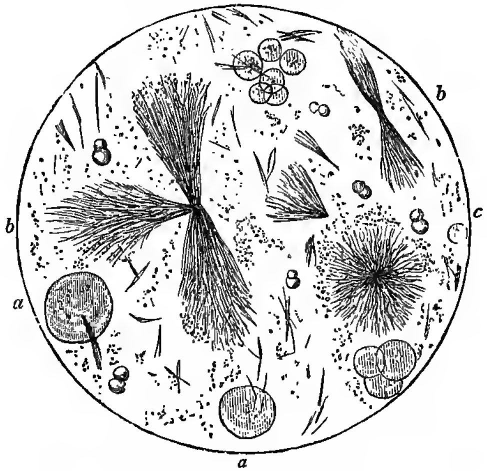


Fig. 22.—(*aa*) Leucin Balls; (*bb*) Tyrosin Sheaves; (*c*) Double Balls of Ammonium Urate.

Urinary concretions of considerable size are occasionally to be seen in urine with the naked eye. They consist, for the most part, of urates, or urates with uric acid. Their recognition is important in the diagnosis of renal colic. When composed of uric acid or acid urates, their color is usually red or brown. Phosphatic concretions of larger size occur more rarely. They are light-colored. Other concretions are occasionally met with.

Foreign bodies occur in the urine from accidental causes, or from negligence in collecting the specimen. We may mention fungi, yeast-cells, micro-organisms, fat globules; fibers of silk, linen, and wool; feathers, wood, starch, etc. Bodies of this kind will be seen in almost every specimen examined. They will not cause any confusion, after a little practice, as they are so different from any of the characteristic urinary sediments that mistakes will rarely be made. Fragments of tumors, as sarcoma, carcinoma, etc., may occasionally be found, and their import is self-evident.

Organized Deposits.—Mucous corpuscles are seen as round, finely granular cells, somewhat larger than blood-corpuscles, and containing from three to five nuclei. They can not be distinguished from the colorless blood-corpuscles. (See Fig. 27.) **Pus-corpuscles** resemble the mucous corpuscles in their appearance. If fresh, warm urine containing mucous or pus-corpuscles be examined under the microscope, the corpuscles will be seen to undergo the ameboid change of shape. When the urine becomes cold, this movement ceases, and the corpuscles assume the globular form. Water causes these corpuscles to swell, the nuclei becoming more distinct, and the outline gradually disappearing. Acetic acid produces a similar change, but more rapidly. Solutions of KOH, NaOH, and NH_4OH disintegrate these corpuscles, destroying the cells and granules; the nuclei

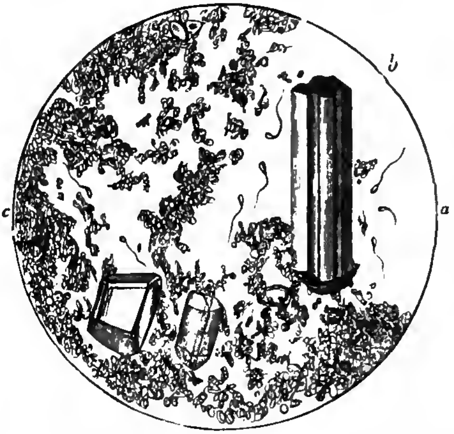


FIG. 23.—(*a*) SPERMATOZOA; (*b*) AMORPHOUS CALCIUM CARBONATE; (*c*) AMMONIUM MAGNESIUM PHOSPHATE.

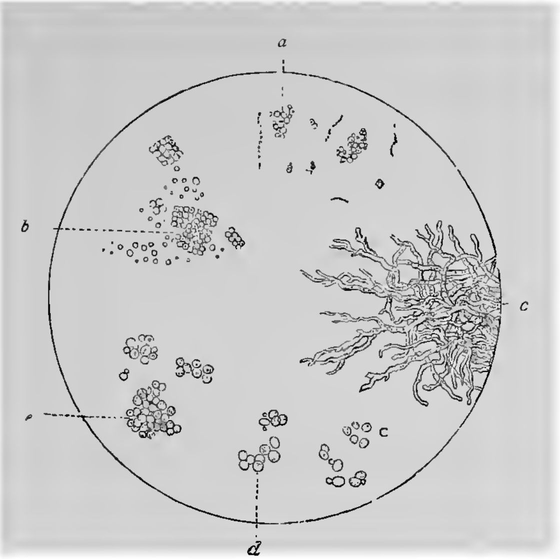


Fig. 24.—(*a*) Micrococci in Short Chains and Groups; (*b*) Sarcinæ; (*c*) Fungi from Acid Fermentation; (*d*) Yeast Cells from Diabetic Urine; (*e*) Mycelium of a Fungus, or Mold.

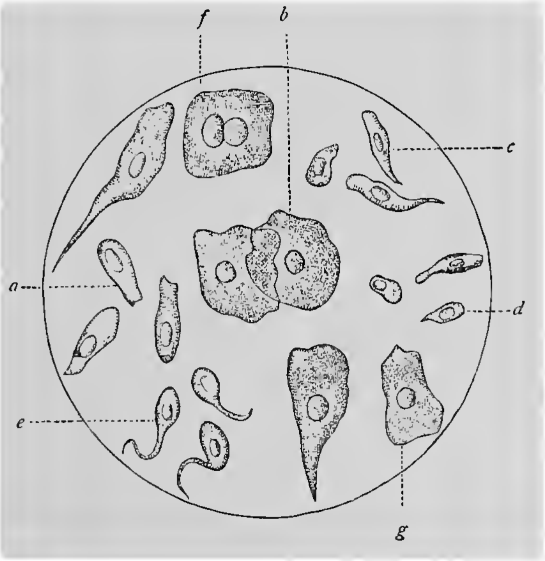


Fig. 25.—(*a*) Epithelial Cells from the Male Urethra; (*b*) from the Vagina; (*c*) from the Prostate; (*d*) Cowper's Glands; (*e*) Littre's Glands; (*f*) Female Urethra; (*g*) Bladder.

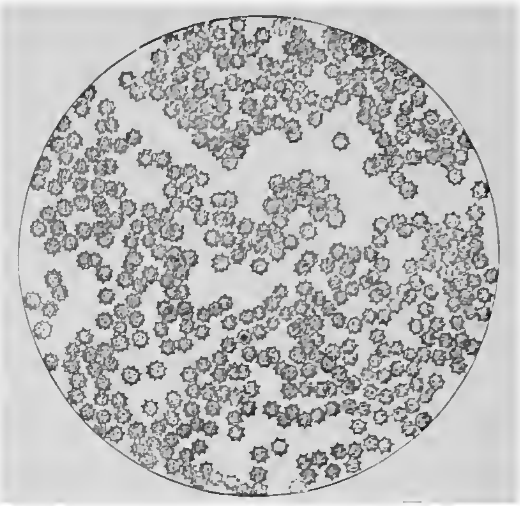


FIG. 26.—CRENATED RED BLOOD-CORPUSCLES IN THE URINE. × 350.

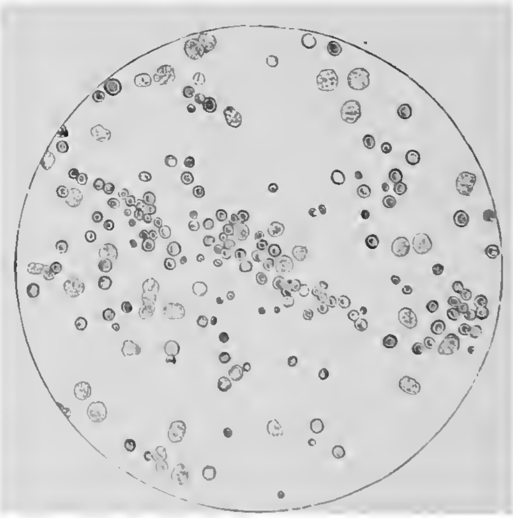


FIG. 27.—RED AND COLORLESS BLOOD-CORPUSCLES OF VARIOUS FORMS.

are the last to disappear. In alkaline urines, therefore, the mucous and pus-corpuscles, if present, rapidly undergo disintegration.

Epithelial cells of a variety of shapes are found in normal urine. Those from the convoluted portion of the tubules are polygonal in shape, but on remaining for some time in the urine they absorb water and become globular. They are a little larger than pus-corpuscles, and may be distinguished from the latter by having but one large, distinct nucleus. The epithelial cells of the loop of Henlé and the larger collecting tubes are columnar in shape. Those from the ureter, pelvis, and male urethra are elongated and conical, containing one nucleus near the center. Large, flat, squamous, epithelial cells are obtained from the bladder, vagina, and female urethra. (Fig. 25.) In chronic cystitis, after the large, flat, irregular cells have been shed off, we may have smaller, rounded cells. Rapidly proliferating cells have a large nucleus in proportion to the remainder of the cell. Old cells, slowly proliferated and desquamated, have a smaller nucleus in proportion to the rest of the cell. This is of importance in the diagnosis of new growths likely to be found in the bladder.

Blood-corpuscles in the urine appear as small, round, disc-shaped corpuscles of a light straw or red color, and when seen on edge appear biconcave. They are prone to changes in form and size on standing for some hours. They undergo decomposition in alkaline urines, change their form, and finally become invisible. (Figs. 26 and 27.)

Casts.—Casts are fibrinous molds of the uriniferous tubules, and frequently contain blood- or pus-corpuscles, epithelial cells, granular matter, crystals, or oil-drops, imbedded in the substance of which they are composed, from which they are named epithelial casts, blood-casts, granular casts, fatty casts, waxy casts, and hyaline casts.

Hyaline casts are perfectly clear, transparent cylinders, without markings, having nearly the same refractive index as the urine, and consequently are not readily seen, especially in a strong light. (Fig. 28.) They are more readily seen with oblique illumination, or by adding a few drops of solution of eosin, Bismarck brown, methyl-green, or fuchsin, to the urine while the sediment is forming. They are characteristic of the very earliest and the recovering stages of nephritis, and are also found in congestion of the kidney, or in simple irritative catarrh of the tubules.

Blood-casts contain blood-corpuscles imbedded in them, and indicate an acute inflammation of the kidney with escape of blood-corpuscles from the circulation into the tubules. (See Fig. 29.) They are characteristic of the very acute stages of nephritis.

Epithelial casts are those in whose surfaces epithelial cells from the tubules are imbedded. (See Fig. 30.) They indicate a rapid shedding of the epithelial cells lining the tubules, and usually occur in the second stage of the inflammation—*i. e.*, when the inflammation has loosened the epithelial cells. They will usually be found only in acute nephritis.

Granular casts are those containing granules, either small or large. The granular matter may come from either the disintegration of the epithelial cells, blood-cells, or from the material of the cast itself. They are frequently spoken of as finely granular, moderately granular, and coarsely granular; the amount of granular matter giving an idea of the amount of the destructive disintegration taking place in the kidney. The dense, coarsely granular varieties, represented by figure 31, *b*, are more especially found in chronic cases. The finely granular cast seen in figure 31, *a*, may be found in the chronic or in the subacute form of the disease.

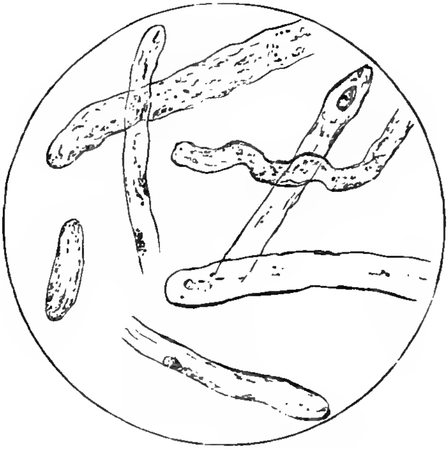


FIG 28.—HYALINE CASTS.

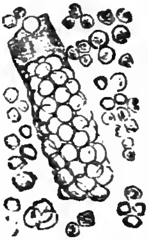


FIG. 29.—BLOOD-CAST.

Fatty casts, or **oil casts**, are such as reveal oil drops in the cast material. They occur in chronic nephritis attended by fatty degeneration. It is sometimes difficult to determine whether the granular degeneration seen in these casts is due to the degeneration of the cast itself, after having been formed, or whether it is the result of the disintegration of blood-cells or epithelial cells. These casts form in the tubules, and often remain there for a considerable time—a sufficient time, perhaps, to undergo granular and even fatty degeneration. It is certain that the coarsely granular and fatty casts are never found in the earlier stages of the disease.

Waxy casts are a variety somewhat resembling hyaline casts, but are more dense and more distorted, and frequently are cracked or torn along the edges, or they have lost the regularity of their outline. They sometimes give a blue color with sulphuric acid and iodine, are more refractive than hyaline casts, and are insoluble in acetic acid, while hyaline casts are soluble.

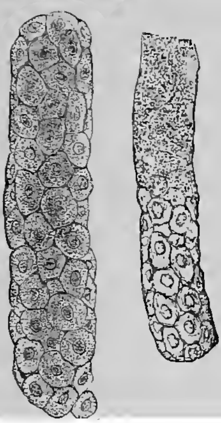


FIG. 30.—EPITHELIAL CASTS.

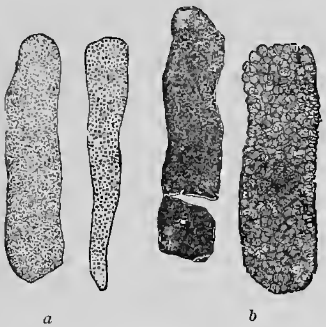


FIG. 31.—GRANULAR CASTS.

Mucous casts are frequently spoken of. They are long, transparent, fibrillar bodies, twisted and branching, and lacking in the terminal features of casts. They should not be regarded as casts, although we may meet with mucous plugs from the follicles in the prostatic urethra which closely resemble casts. The character of the epithelial cells, with which they are associated, will usually serve to distinguish them. The absence of albumin will also assist. True renal casts without albumin in solution are rare. Hyaline casts without albumin are not rare, but they are frequently mucous instead of true tube-casts.

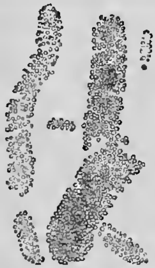


FIG. 32.

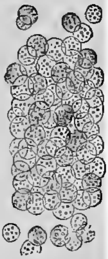


FIG. 33.

Casts can usually be readily distinguished from other bodies met with in the urine by the peculiarly rounded end, formed by the pushing of the cast material through the tubule by pressure from behind, while still in a plastic condition. This rounded extremity is one of the most characteristic features in casts, and when in doubt as to the identity of an object, this will often serve as a guide.

Casts like formations of **urates** will occasionally be met with, and always resemble granular casts. (See Fig. 32.) Masses of micrococci closely resembling casts will also occasionally be seen, but these can usually be distinguished by their appearance, or by their resistance to reagents, as caustic potash, nitric acid, etc. **Leucocyte casts** (Fig. 33) are met with in suppurating conditions of the tubules of the kidney, in gonorrhea, prostatitis, and leucorrhea.

Granular Detritus.—Under this name we will designate the ill-defined granular or disintegrating masses of material frequently met with in cases of nephritis. These irregular or amorphous masses are probably partially disintegrated cells or masses of free granules of this origin. The amount of this material in any specimen of nephritic urine should be noted, as an aid in arriving at a clear idea of the amount of destructive change going on in the kidney. This point is an important one in prognosis, as by it we are able to determine that organic destruction of the kidney is rapidly progressing and the prognosis unfavorable; or, that there is little or no organic destruction and the prognosis better.

The following scheme for the chemical and microscopical examination of sediments will be found useful as a guide to rapid work:

URINARY DEPOSITS.

CHEMICAL EXAMINATION.

Draw off a portion of the sediment with a pipette or glass tube, and transfer to a watch-glass or small test-tube.

White Deposit.	Dissolves on heating urine,			*Ammonium urate.*
	Insoluble on heating.	Sol. in NH_4OH,		*Cystin.*
		Insol. in NH_4OH,	Soluble in acetic acid,	*Earthy Phosphates.*
			Insoluble in acetic acid,	*Calcium oxalate* or *oxalurate.*
		Gelatinizes in NH_4OH,		*Pus* (see above).
Colored Deposit.	Visibly crystalline (red),			*Uric acid.*
	Amorphous,	Pale, easily soluble by heat,		*Urates*
		Deep-colored, slowly soluble by heat,		*Acid urates with uroerythrin.*
		Red, insoluble by heat, alkalies, or acids,		*Blood.*

MICROSCOPICAL EXAMINATION.

With a clean pipette draw off a small portion of the sediment, transfer to a clean glass slide, and examine with a ½-in. or ¼-in. objective. A cover-glass may be dispensed with.

Deposit is Amorphous.		Small granules with spicules on larger granules; vanishes on adding KOH or NaOH,	light = *Sodium urate.* dark = *Ammonium urate.*
		Permanent on adding KOH or NaOH,	*Calcium carbonate* (rare).
		Globules, strongly refracting light,	*Fat.*
Deposit is Crystalline.	Urine, Acid.	Yellow, cross or whetstone shaped, or in groups,	*Uric acid.*
		Regular octahedra, envelope-shaped,	*Calcium oxalate.*
		Hexagonal plates, soluble in NH_4OH (white),	*Cystin.*
		Bundles of needles crossing each other,	*Tyrosin.*
	Alkaline Urine.	Large prisms, soluble in acetic acid (coffin-lid shaped),	*Ammonium magnesium phosphate.*
		Brown, double spheres, spiculated,	*Urate of ammonium.*
		Club-shaped crystals, single or in groups,	*Calcium phosphate.*
		Double spheres, radiated structure, soluble in acetic acid, with effervescence,	*Calcium carbonate* (rare).
		Double spheres, insoluble in acetic acid,	*Calcium oxalurate* (rare).
		Double spheres, yellow or red, striated,	*Uric acid.*
Cellular Elements.		Red or yellow discs, biconcave; sometimes irregular in outline,	*Blood-cells.*
		Granulated corpuscles. With diluted acetic acid, show 3 to 5 nuclei,	Albumin present, *Pus.* Albumin absent, *Mucous corpuscles.*
		Round, conical, or flat cells with one nucleus,	*Epithelium from urinary tract.*
		Tadpole-shape, with long tail (small),	*Spermatozoa.*
		Cylinders, parallel margins, clear, granular, or containing epithelial cells or blood-cells,	*Casts of uriniferous tubules.*
		Fungi, yeast, hairs, threads, etc., etc.,	*Extraneous matters.*

URINARY CALCULI.

Urinary calculi, or concretions, are hard masses of urinary sediments formed in the kidney, ureters, bladder, or sinuses of the prostate gland. They are **simple,** composed of one kind of material, or **compound** or **mixed,** composed of two or more kinds of material, deposited in concentric layers. In the examination of a calculus it should be sawed through so as to expose these layers, and small portions of each layer examined separately. An examination of a calculus is necessary to determine the condition which led to its formation, and to suggest proper treatment to prevent the formation of others. About sixty per cent. of all urinary calculi are composed of uric acid or acid urates. They are generally reddish and smooth, but sometimes tuberculated. About forty per cent. of the remainder of the stones are mixed uric acid and earthy phosphates, containing rather more of the latter ingredients. When the calculus starts as a uric acid concretion, and the urine changes from acid to alkaline, the phosphates are deposited. This is apt to occur sooner or later. The cross-section of such a calculus shows very plainly the different layers.

Calcium oxalate calculi, or **mulberry calculi**, comprise about three per cent. of all cases operated upon. They are gray or dark brown, very hard, and generally tuberculated, when they are often called "mulberry calculi." When smooth they are often called "hempseed calculi."

The phosphatic calculi are rare, as are those composed of calcium carbonate, cystin, xanthin, fibrin, blood, indigo, and urostealith.

The following is a scheme for the qualitative examination of calculi:

These last, composed of calcium and magnesium soaps, fat, albumin, etc., are very rare.

Heat a portion of the powdered stone on a platinum-foil or charcoal with blowpipe.

A. *It chars and burns with a flame.* Probably xanthin, cystin, urostealith, or fibrin.

1. The flame burns briefly, emitting odor of SO_2. The powder dissolves in ammonia, and on diluting deposits six-sided plates = **Cystin.**

2. It does not give the murexid test. The powder is soluble in HNO_3, without effervescence, and the dried residue becomes orange with alkalies and red on warming = **Xanthin.**

3. The flame is yellow, prolonged, and gives the odor of burning shellac. The powder is soluble in alcohol = **Urostealith.**

4. The flame is yellow, prolonged, and gives the odor of burning feathers. Soluble in hot KOH solution, and is precipitated again by acetic acid = **Fibrin.**

B. *It chars, but does not burn with flame.*

1. The powder gives the murexid test.

a. It gives off NH_3 when warmed with KOH solution = **Urate of Ammonium.**

b. It gives no NH_3 with KOH = **Uric Acid.**

C. *The powder does not strongly char or burn.* Treated with diluted HCl.

1. It dissolves with effervescence = **Calcium Carbonate.**

2. It dissolves without effervescence, and the solution gives a white precipitate with NH_4OH = **Phosphates or Calcium Oxalate.**

Treat the powder with acetic acid.

Phosphates dissolve without effervescence.

Mixed phosphates fuse in heating on foil.

Calcium phosphate does not fuse.

Triple phosphate gives off NH_3 when warmed with a little KOH solution.

Calcium oxalate is insoluble in acetic acid. After ignition it gives an alkaline powder, which effervesces with acetic or diluted HCl.

The subjoined table gives the most prominent variations in physical and chemical characters, with brief notes of their significance. As there are numerous handbooks upon this subject, the student is referred to them for details:

THE URINE OF THE TWENTY-FOUR HOURS—NORMAL AND PATHOLOGICAL.

PHYSICAL CHARACTER.	NORMAL.	ALTERATIONS IN ABNORMAL CONDITIONS.
Color.	Pale straw to reddish yellow. The average color is amber.	Colorless: neuroses, chronic nephritis, diabetes. High-colored: acute fevers, icterus. Blood-red: blood or foreign color. Dark brown: hematuria, poisoning by carbolic acid, potass. chlorate, or iodoform. Smoky brown: presence of decomposed blood, acute nephritis. Yellow or green: presence of bile. White: chyle or pus.
Transparency.	Clear, with only a slight flocculent cloud of mucus.	Urine turbid when passed, is abnormal. Whitish sediment may be pus, phosphates, or ammonium urate.
Consistence.	When normal, urine is mobile, like water.	When viscid, it indicates albumin, bile, mucus, or pus.
Odor.	Peculiar to itself.	Urine putrid when passed, indicates cystitis.
Reaction.	Slightly acid; becomes more acid on standing, then becomes alkaline.	Urine strongly acid in fevers and inflammations of the liver, heart, and lungs; in acid dyspepsia. Urine is alkaline in cystitis, and occasionally in debility, chlorosis, certain organic nervous diseases, typhus, etc.

THE URINE OF THE TWENTY-FOUR HOURS—NORMAL AND PATHOLOGICAL.—*Continued.*

CONSTITUENTS.	AMOUNT IN GRAINS.	ALTERATIONS IN PATHOLOGICAL CONDITIONS.
Sulphuric acid.	23 to 38	Having more or less the same source as urea, it will increase or diminish therewith. Occurs as sulphuric ethers, and preformed.
Phosphoric acid.	46 to 54	**Increased** in fevers, in nerve-exhaustion, disease of spinal cord, and in tubercle of the lung. In phosphatic diabetes the alkaline phosphates are greatly increased. **Diminished** in many mental diseases, especially mania, and in chlorosis.
Oxalic acid.	0.3	**Increased** in catarrhal jaundice, and in oxalic acid diathesis, mental depression, and certain forms of dyspepsia.
Phosphate of lime.	4 to 5	**Increased** in osteomalacia, rickets, scrofula, carcinoma, long-continued suppuration, organic disease of the spinal cord. **Diminished** in fevers.
Phosphate of magnesium.	7 to 11	
Chloride of sodium.	150 to 200 Cl = 90 to 120 Na = 60 to 80	**Increased** in fevers at the outset, and with the reabsorption of dropsical fluids. **Diminished** during apyrexia, dropsies, cholera, typhus, inflammations generally, and especially in the forming stage of pneumonia.
Free acid (calculated as oxalic acid).	30 to 60	**Increased** during the acme of acute febrile affections (on account, probably, of the diminished proportion of water present). **Diminished** in most diseases affecting the nutrition and leading to a deficiency thereof.
Total inorganic salts.	200 to 380	
Potassium.	38 to 48	
Sodium.	140 to 180	
Calcium.	4 to 5	
Magnesium.	2 to 3	

ORGANIC CONSTITUENTS.

Urea.	450 to 500 or 30 to 34 gm.	**Increased** after much meat, fevers, diabetes mellitus, copious drinking of water or alcohol, congestion of liver. **Diminished** in abstinence from meat, rest, hepatic abscess, nephritis, chronic wasting diseases.

THE URINE FOR THE TWENTY-FOUR HOURS—NORMAL AND PATHOLOGICAL.—*Continued.*

ORGANIC CONSTITUENTS.	AMOUNT IN GRAINS.	ALTERATIONS IN PATHOLOGICAL CONDITIONS.
Uric acid.	4 to 15 (ratio to urea, 1 : 40)	Increased leucocythemia, pernicious anemia, gout, rheumatism, deficient oxidation; organic diseases of heart, lungs, liver, or skin; after acute fevers and excessive meat diet. Diminished in vegetable diet, gout, before attack, chronic renal disease, outdoor exercise.
Hippuric acid.	5 to 15	Increased with vegetable diet, after taking benzoates. Decreased in animal diet.
Creatinin.	8 to 15	Increased in meat diet, and increased nitrogenous metabolism. Decreased in vegetable diet, and milk diet.
Xanthin Bases.	0.5 to 2	Increased in splenic disease, meat diet, deficient oxidation. Decreased in vegetable diet.
Carbolic acid, Cresol, etc.	0.015	Increased in certain diseases of the intestines, causing constipation (ileus, etc.), but has been observed to be increased also in certain cases of diarrhea.
Indoxyl.	0.07 to 0.05	Increased with diseases attended by constipation and intestinal fermentation, and occasionally, also, in cases of diarrhea. After cholera, cancer of the liver and stomach, purulent peritonitis. Valuable diagnostic sign in typhoid fever and cancer of the liver.
Acetone, Diacetic acid, Hydroxybutyric acid.	Traces. " "	Increased in diabetes mellitus; conditions of increased proteid metabolism, with deficient oxidation.
Albumin.	None.	Nephritis, pregnancy, poisoning by certain substances, cold baths, violent exercise, rheumatism, infectious fevers, etc.
Albumose.	None.	Presence not clearly diagnostic.
Peptone.	None.	" " " "
Dextrose.	None or trace.	Glycosuria and diabetes mellitus.
Lactose.	None.	During lactation, after weaning.
Bile.	None.	Obstruction in bile-duct, structural hepatic diseases, malaria, pernicious anemia, yellow atrophy of liver, typhoid fever, and AsH_3 poisoning.
Blood.	None.	Hemorrhages, giving hematuria; hemoglobin in malaria, acute nephritis (sometimes).
Pus.	None.	Suppuration.
Mucus.	Present.	Increased in any irritation along the urinary tract, by uric acid, calcium oxalate, etc., catarrh of bladder, urethra, vagina. Usually increased in acute fevers.

NOTES ON URINARY DIAGNOSIS.

In order to clearly understand the diseases of the kidney and the variations in the quantity and quality of the urine in health and disease, the student should study the minute anatomy and the physiology of the kidney.

It is not within the province of this book to teach physiology, and the student is urged to consult some work on physiology for information upon this subject.

It is sufficient here to remind the student that the kidney has a double capillary circulation. The blood enters the kidney by the renal artery under an estimated pressure of about 2½ pounds to the square inch. The renal artery, after entering the kidney, rapidly divides into many small branches, each of which abruptly terminates in a glomerulus, or ball-like tuft of short capillaries, inclosed in a capsule known as the capsule of Bowman. This capsule is the upper end of a tube for collecting the urine. The veins, after leaving the glomeruli, pass downward toward the medullary portion of the kidney, rapidly dividing and anastomosing with other similar veins, making a closely woven network of capillaries about the convoluted portion of the tubules containing the fluid filtered into them at the upper end, or in the capsule. Immediately below the capsule the tubules become very much contorted, and are lined with thick, polygonal, glandular-like epithelial cells. At the border between the cortical and medullary portions of the kidney, the tubes become straight and narrower, forming a loop dipping well down into the latter and returning to the former. The returning limb of this loop is about three times as large as the descending limb. This loop of Henle is lined with clear, flattened, epithelial cells, with prominent or bulging nuclei, as though these nuclei were intended to retard the current of fluid within the tube. As the tube returns to the cortical portion of the kidney the epithelial cells become thicker, and assume the glandular and cuboid appearance, while the tube again becomes convoluted or spiral. These tubes then combine into larger, straight tubes, called collecting tubes, which pass downward toward the hilum and discharge their contents into the pelvis of the kidney. The structures above described, with nerves and lymphatics, are held together by an abundant supply of loose connective tissue, except in the medullary portion, where it is quite firm and compact. This connective tissue is especially abundant along the arteries and veins, and seems to be continuous with the membrane or capsule covering the whole kidney.

QUANTITY OF URINE.

The volume of liquid secreted by the kidneys is largely proportional to the pressure brought to bear on the plexus of arterioles of the glomeruli. The glomerulus is subjected to greater pressure than other capillaries in the body. The urine is composed of water and certain solids held in solution, with usually certain sediments in suspension. Some writers regard the kidney as merely a filter, others as a dialyzer and filter, while others look upon it as a compound organ. They look upon the glomerulus as a filter, and the epithelium of the convoluted tubes as a gland similar to the other glands, as the parotid, etc. Clinical observation, as well as experimental physiology, shows that the quantity of water is principally dependent upon blood pressure, and in this respect the kidney acts as a filter. The more active the circulation, the more active the secretion of urine. We must not forget, however, that the injection of certain substances, such as glucose, urea, caffein, etc., into the circulation of an animal increases the flow of water.

The local blood pressure in the glomerulous tufts may be increased : (1) By an augmentation of the general blood pressure, by an increase in the force or frequency of cardiac systole, or by an increased resistance to the flow of blood in other vascular areas. (2) By a relaxation in the renal artery, which will allow the full force of the blood stream to impinge on the capillaries of the Malpighian tufts. This relaxation must, of course, not be attended by a general relaxation, as this lowers the general blood tension. Digitalis increases the blood pressure, and hence acts as a diuretic. Strophanthus, while stimulating the heart, does not materially increase blood pressure, and hence is not so effective as a diuretic.

The nitrites, when given in not too large doses, dilate the renal artery, and thus increase the flow of urine. Cold constricts the capillaries in the skin, and thus throws more blood into the kidneys and increases the urine.

A diminished blood pressure in the glomeruli may be produced by : (1) A general lowering of the blood pressure by a diminution of the force and frequency of cardiac systole. (2) A general dilatation of the capillaries all over the body, or in some large area other than the kidney. Belladonna and heat dilate the capillaries, lower blood pressure, and lessen the flow of urine. (3) By a constriction of the renal artery, thus shutting off the current of blood from the tufts.

These general facts help us in understanding many of the physiological as well as pathological variations in the quantity of urinary secretion. These principles have been well verified by experiment and clinical observation. All physiologists admit that the pressure that the glomeruli are subjected to is enough to cause osmosis or filtration.

We must remember, too, that the urine is also governed, both in quantity and quality, by food and drink. It is varied by exercise and sleep, heat and cold, emotions and alcohol. We should not forget that there is a complementary action between the kidneys and skin on the one hand, or the intestine on the other. Perspiration and diarrhea may assist the kidneys, or, more properly, do part of their work.

The kidney is not alone a filter, separating water holding a few salts in solution ; however, if we submit urine to filtration, it all passes except certain accidental substances, as mucus, epithelial cells, etc., added to it along the tract below the point where it is secreted. So far as the composition of the urine is concerned, there is nothing to militate against the theory that the function is merely that of a filter. When we look at the blood, however, we find a solution of albumin, urea, salts, etc., which, under pressure, may be filtered through animal membranes. Probably we may accept 2½ pounds per square inch as the pressure of the blood in the renal tufts. Under this pressure albumin will filter through dead animal membrane. We must, then, so far as albumin is concerned, suppose that the kidney has an elective action on the blood. The structure of the kidney indicates a double process. The second set of capillaries seems to be intended to furnish nourishment to the tubules. Moreover, in the amphibia (the newt) there are two separate blood supplies to these two sets of capillaries: the renal artery, supplying the glomerulus, and a branch of the femoral vein, supplying the second set of capillaries. Nussbaum tied the artery supplying the glomeruli, and saw the urine cease to flow. When the femoral vein was tied, the urine continued to flow. The injection of substances into the circulation, while one of these vessels was tied, allowed him to determine what substances were secreted from each set of capillaries. He found that water. peptone, egg-albumin, and sugar were secreted before ligating the renal artery,—*i. e.*, by the glomeruli,—but that they disappeared when this operation was performed. Urea, however, continued to appear. On ligating the vessel that supplied the second set of capillaries, the urine almost disappeared until the urea was injected, when the water increased, probably to dissolve the urea. On injecting indigo-carmine no urine was secreted, but the indigo was found in the tubular epithelium. This seemed to prove that urea is excreted by the epithelium of the tubules.

Heidenhain's experiments prove that indigo is separated from the blood by the tubular epithelial cells.

The glandular character of these epithelial cells seems to point to their use in performing some office other than that of a filter.

If a section of the spinal cord of an animal be made below the medulla, there is almost total suppression of urine from decreased blood pressure. If urea or uric acid be injected into the blood of such an animal, there is a copious flow of urine. The explanation of this is probably their stimulation of the epithelial cells. Bile pigments, the organic salts of iron, and fat of milk, when injected into the circulation, are secreted by the tubular epithelium. Ludwig holds that blood-serum, minus the proteids and fats, is secreted in the capsule. This washes out the tubules and is largely reabsorbed, leaving the solids in denser solution. Ribbert showed that the urine secreted by the cortex was more watery than the whole urine. He extirpated the medullary portion of the kidney of a rabbit and collected the urine of the cortex. It was watery. He thinks, with Ludwig, that water is reabsorbed in Henle's loops where the tube is smallest. The tortuous collecting tubes are to retard the current, to allow time for absorption. Many physiologists claim that albumin filters through the capsule and is reabsorbed in the tubules. Bowman, Heidenhain, Von Wittich, and Danders claim that water and inorganic salts filter through the capsule, and urea and uric acid are separated by the epithelial cells of the tubules.

SPECIFIC GRAVITY.

(See p. 632.)

Closely associated with the question of quantity, and often dependent upon the latter, is that of specific gravity. Other things being equal, the specific gravity and quantity are inversely proportional to each other—*i. e.*, the specific gravity decreases as the quantity increases, and the reverse. In drawing conclusions from the specific gravity, the quantity passed in twenty-four hours must always be considered. Causes which diminish the quantity of water must raise the specific gravity, and those which increase the water diminish the specific gravity, provided the kidney is performing its secretory functions. In drawing conclusions based upon these two data, it is necessary to make more than one observation, if possible, as a temporary condition will call for a different diagnosis from that of a permanent variation from the normal. With proper precautions, these two factors furnish information of the greatest importance to the diagnostician.

Most urinometers are graduated to give the specific gravity marked on the stem, at 15° C. (60° F.). Warm urine is lighter than cold urine. When the temperature of the urine is above 15° C. (60° F.), a correction should be made by adding one degree to the observed gravity on the urinometer for each 3° C. (5.4° F.) above that temperature. When the temperature is below 60° F., a similar allowance must be made and deducted from the reading.

Thus, if the specific gravity at 75° F. were found to be 1016, the corrected reading would be:

$$75 - 60 = 15. \qquad 15 \div 5.4 = 2.8. \qquad 1016 + 2.8 = 1018.8.$$

This rule is not quite exact, but near enough for clinical purposes.

Specific gravity is of use in calculating total solids. (Compare p. 634.) The following table gives the number of grains of solid matter in a fluidounce of urine of the given specific gravity:

Specific gravity of		1005	gives	5.10	grains	of solids in	1 oz.
"	"	1010	"	10.20	"	"	1 oz.
"	"	1015	"	15.30	"	"	1 oz.
"	"	1020	"	20.40	"	"	1 oz.
"	"	1025	"	25.50	"	"	1 oz.
"	"	1050	"	51.00	"	"	1 oz.

The amount of solid matter excreted in twenty-four hours by an average adult man is about 60 to 65 gm., or 1000 grs. From the above table it is seen that the last two figures of the specific gravity very nearly express the number of grains in each fluidounce of normal urine. By multiplying these two figures by the number of ounces passed in twenty-four hours, we obtain approximately the number of grains of solid matter passed in twenty-four hours. Various attempts have been made to determine a coefficient or formula to give the result in grams per liter. Trapp obtained the number 2; Loebisch, 2.2; Christison and Haeser, 2.33. Thus, if a given urine has a sp. gr. of 1020, it will contain, approximately, $20 \times 2.2 = 44$ gm. per liter. If the quantity of this same urine be 1200 c.c. per twenty-four hours, then the total solids will be found by multiplying the above result by the number of liters— $44 \times 1.2 = 52.8$. The most striking variations from the normal solids will be found in diabetes mellitus and in the various forms of nephritis.

Urine is pathological if we observe:

1.	Specific gravity	high,	color	high,	quantity	small.
2.	" "	"	"	pale,	"	large.
3.	" "	low,	"	high,	"	large.
4.	" "	"	"	very pale,	"	small.

If urine is persistently below 1015, suspect albuminuria and nephritis; if below 1008, suspect albuminuria or hydruria. If persistently above 1025, color pale, quantity increased, it indicates diabetes. If persistently above 1025, color high, quantity diminished, suspect fevers, some cardiac weakness, or excess of uric acid.

Examples.

CASE 1.—Quantity in twenty-four hours, 900 c.c. ($\frac{9}{10}$ of a quart); specific gravity, 1030 to 1035; color high; severe fever.

CASE 2.—Quantity, 4800 c.c. (5 quarts); specific gravity, 1025 to 1035; diabetes mellitus.

CASE 3.—Quantity, 3500 c.c. (3½ quarts); specific gravity, 1015; diabetes insipidus.

CASE 4.—Quantity, 600 c.c.; specific gravity, 1010; chronic nephritis.

Reaction.—The normal reaction of urine is feebly acid—*i. e.*, it reddens blue litmus paper. (Compare p. 632.)

The cause of the acidity of the urine is believed to be chiefly the presence of acid sodium phosphate, with minute amounts of carbonic, hippuric, and lactic acids, or their acid salts.

The urine occasionally becomes neutral or alkaline for a short time after meals, or after cold baths.

The acidity of the urine is increased:

1. In concentrated urines.
2. After prolonged exercise.
3. After meat diet.
4. Acute fevers; rheumatism, scarlatina, etc.
5. Gout, diabetes, scurvy, acid dyspepsia.

The acidity is diminished or is alkaline:

1. After a purely vegetable diet.
2. After taking alkaline salts.
3. Anemia, simple and pernicious, and hypochlorhydria.
4. Alkaline fermentation; cystitis.

Color.—The color of normal urine is amber. Diluted urine varies from straw-color, to colorless. Concentrated urine is orange or reddish-yellow. Febrile urine, reddish-brown. Bloody urine is red. The color is smoky in acute nephritis, after $KClO_3$, C_6H_5OH, creosote, AsH_3, etc. Yellow or green indicates bile, icterus. A dirty blue color is sometimes seen in cholera, typhus, and from indican. A white color may be due to chyle, pus, phosphates.

Effects of Drugs, etc.—Dark: C_6H_5OH, creosote, $KClO_3$, tar, etc., turpentine, terpin hydrate, salol, quinine (sometimes), phenocoll, naphthalene, antipyrin. Red: Analgen, cascara, sulphonal (sometimes violet-red), madder, aloes, and magenta. Yellow: Thallin, santonin, cascara, rhubarb, senna, chrysophanic acid, and gamboge. Blue: Indigo, methylene blue, and pyoctannin.

INORGANIC CONSTITUENTS OF THE URINE.

The most important inorganic constituents of urine, from a diagnostic point of view, are the chlorides, phosphates, and sulphates. We may include among these, for convenience, calcium oxalate, although the acid is organic. (Compare p. 635.)

Chlorides.—Daily amount, ten to fifteen grams (150 to 200 grs.).

Increased:
1. After muscular exercise.
2. After taking much food or drink.
3. During absorption of exudates.
4. In acute Bright's disease, diabetes insipidus, and ague, after chill.

Diminished:
1. In all acute fevers, especially acute pneumonia. (Increases after crisis.)
2. During formation of exudates and dropsies.
3. In chronic nephritis, certain dyspepsias, diarrheas, cholera, typhoid.

Phosphates.—Daily amount of phosphoric acid, two to four grams (30 to 60 grs.).

Increased:
1. After mental work, hysteria, certain nervous diseases, or worry.
2. After muscular exertion, meat diet, copious drinking of fluids.
3. Rachitis, osteomalacia, early phthisis, diabetes mellitus, phosphatic diabetes, some fevers.

Diminished:
1. Most acute diseases, renal diseases, rheumatism, gout, pregnancy.

Sulphates.—Daily amount of sulphuric acid, 1.5 to 3 grams (22 to 45 grs.). (See p. 637.)

Sulphuric acid occurs as preformed and as ethereal sulphates. The latter is treated under the pigments, page 126. Little diagnostic value is attached to variations in the preformed sulphates.

Increased:
1. Meat diet, prolonged exercise, after taking sulphur compounds.
2. Fevers, rheumatism, pneumonia, delirium tremens, cerebral meningitis.

Diminished:
1. Vegetable diet (usually).
2. Nephritis, chlorosis, chronic diseases generally.

ORGANIC CONSTITUENTS OF URINE.

(See p. 638.)

Formation of Urinary Constituents.—This has been the subject of a great deal of discussion. Do they exist preformed in the blood, or are they prepared in the kidney by the epithelial cells? Urea is the chief nitrogenous portion of the urine. Urea is formed outside the kidney, and is found in the blood. (1) Blood contains 1 in 3000 to 5000. The renal vein contains less than the renal artery. (2) After nephrectomy in animals, the urea increases gradually to 1 in 300 to 400. At the same time, diarrhea and vomiting come on. The fluids so voided contain urea. Such animals die in one to three days after operation. (3) After ligature of ureters, the urine ceases and urea accumulates in the blood. Urea is not formed in the muscles or nerves, but in the liver. Hoppe-Seyler denies that the liver contains urea. But Gescheidlen, Cyon, Von Schroeder, and Minkowski affirm its production in the liver. They find that the urea in the blood increases in passing through the liver. By ligating the portal vein in geese, Minkowski found that lactic acid replaced uric acid in stools of this bird. Parkes noticed that in hepatic abscess, in the congestive stage, urea is increased; in suppurative stage it is diminished. Urea is diminished in cancer of the liver, phthisis, and cirrhosis (usually). In congestion it is increased. Bruising the liver increases the excretion of urea, because of the resulting congestion. In acute yellow atrophy of the liver the urea is greatly decreased or absent, while leucin and tyrosin, the antecedents of urea, take its place. Urea is decreased in phosphorus poisoning. Drugs that increase bile (hepatic activity) also increase urea—viz., sodium benzoate, sodium salicylate, colchicum, $HgCl_2$, Hg_2Cl_2, and euonymin. Uric acid is formed outside of the kidney, and especially in the liver and muscles. In certain disorders of the liver and skin there is an excessive elimination of uric acid. It is increased in phthisis, and in most diseases in which oxidation is interfered with, in many or most febrile conditions, and in diseases of the lungs or heart. It is increased by all diseases accompanied by leucocytosis, or increase of white blood-cells, as in **scurvy, leucocythemia, pernicious anemia,** and during the **infectious** or **specific fevers.** (See Uric Acid, p. 466.) For the origin of the coloring matters, xanthin bases, creatinin, etc., the student is referred to these subjects in part IV, page 468, *et seq.*

Hippuric acid is formed partly, at least, in the kidney. If a solution of sodium benzoate and glycin in blood be passed through the blood-vessels of a freshly excised kidney, hippuric acid appears in the blood issuing from it. The reaction of kidney tissue is always acid, even in those animals which secrete alkaline urine.

Urea.—We have already discussed the formation of urea in the body, with the conclusion that it is principally formed in the liver. As we have stated, about 85 to 88 per cent. of the nitrogen from tissue metabolism in the body escapes by the urine as urea; hence we may look upon the amount of urea eliminated as a measure of tissue change. This is only true in health. In certain diseases—as, for example, in yellow atrophy of the liver—the urea may almost disappear from the urine, and the nitrogen will escape in some other form. When we wish to measure the tissue metabolism, we usually determine the total nitrogen eliminated by the kidneys. This will give the amount of proteid metabolism going on in the body. It is usual to express the nitrogen as urea. One hundred gm. of muscle contain about 3.4 gm. of nitrogen, corresponding to 7.286 gm. of urea. Or, 1 gm. of urea is equivalent to 13.72 gm. of muscle tissue, and 1 gm. of nitrogen is equivalent to 29.4 gm. of muscle tissue. If we know the amount of nitrogen taken as food, and the amount excreted by the urine and by the feces, we can calculate just what is going on in the body. When the amount of intake and excretion of nitrogen are equal, the body is said to be in nitrogen equilibrium—*i. e.*, it is neither gaining nor losing nitrogen. As the amount of urea must vary with the amount of nitrogen ingested, a mere statement of the amount of urea excreted in twenty-four hours, without taking into account the amount of nitrogen taken as food, can not mean very much. The nitrogenous metabolism must be more in a large, robust man than in a feeble, half-fed one. It must be more in a man of 200 pounds than in one weighing 125 pounds. According to Uhle, adults pass, in twenty-four hours, for each pound of body-weight, 0.17 to 0.27 gm. of urea. A man of 150 pounds, according to these figures, would pass from 25.5 to 45 gm. of urea in each twenty-four hours; while a man weighing 200 pounds would pass from 34 to 50 gm. We may formulate this fact in this manner: For every 100 pounds in body-weight, an adult ought to excrete about 20 to 25 gm. of urea in each twenty-four hours. Children excrete more in proportion to their body-weight than adults. Thus, from three to six years of age, they secrete for every pound of body-weight 0.45 gm. of urea in each twenty-four hours; at eight to eleven years, 0.36 gm.; at thirteen to sixteen years, from 0.18 to 0.27 gm. in twenty-four hours.

The Relation between the Excretion of Urea and Chlorides.—In normal conditions the chlorides, calculated as NaCl, are about one-half as much as the urea. When the urea is being derived from the destruction of tissue, or when the body is living upon its own proteids, this ratio will be changed. For the tissues contain much less NaCl than our average food. In total abstinence from food, the NaCl almost disappears from the urine. In all febrile diseases, we should expect that this ratio would be disturbed, as it usually is.

The excretion of urea is increased in all acute fevers. In such cases the patient eats little, and excretes more urea than in health, and less chlorides. This ratio between urea and chlorides may sometimes assist us in diagnosis of certain obscure wasting diseases. In acute febrile diseases the excretion of urea has the following course: At the beginning, and until the acme is reached, urea is increased to 40, 50, or even 60 gm. in twenty-four hours. It then grows less, and falls below normal as the fever abates, and gradually comes up to normal again during convalescence. In intermittent fever the urea is increased before the chill or paroxysm.

In those febrile diseases which end by a crisis, as pneumonia, the increased elimination of urea may continue for several days after the crisis. Another exception to the general course of the elimination of urea is in the disease known as acute yellow atrophy of the liver. In this disease the urea nearly or entirely disappears from the urine, and is replaced by leucin and tyrosin. In most chronic diseases with diminished nutrition, it is below normal. In hectic exacerbations it increases during the fever. Near the fatal termination of fevers the urea decreases very much, or it may almost disappear during the last twenty-four hours. Among the non-febrile diseases in which urea is increased, the most marked case is diabetes mellitus, in which it often rises as high as 100 to 150 gm. in twenty-four hours. A partial explanation of this large amount of urea is found in the large amount of food taken by such patients, although there is an increased tissue-destruction in this disease. A moderate increase has been noticed in cases of pernicious anemia, severe cases of leucocythemia, scurvy, chorea, and paralysis agitans. There is usually an increase after epileptic seizures, and some have noticed the same thing after attacks of hystero-epilepsy. **Certain drugs cause an increased elimination of urea.** An increase is produced by coffee, caffeine, morphine, ammonium chloride, NaCl, KCl, Li_2CO_3, and the ingestion of large drafts of water. In regard to the effects of quinine, salicylic acid, and cold baths, opinions differ. It is greatly increased in phosphorus poisoning. Electrical stimulation seems to increase the elimination of urea. **The elimination of urea is decreased** by any cause that interferes with hepatic activity, as in all organic diseases of this organ. It is very much decreased in carcinoma and cirrhosis of the liver, and in acute febrile jaundice (Weil's disease). A careful estimation of the output of urea is then often of service in the diagnosis of hepatic disease. In chronic lead poisoning urea is decreased. Also in Addison's disease. In most forms of nephritis, especially in all forms in which the tubules or the circulation are affected, urea is diminished. As urea is separated by the epithelium lining the convoluted tubes, any disease of this epithelium will interfere with its excretion.

Uric Acid.—$C_5H_4N_4O_3$. Uric acid is found as a normal constituent of the urine of all carnivorous animals and of birds. This acid was formerly regarded as an antecedent of urea. This idea is now abandoned by most physiologists. The origin of uric acid has been the subject of much discussion and experimentation. The latest notion of the generation of urea and uric acid regards leucin, tyrosin, glycocol, and asparaginic acid as the antecedents of urea, and nucleinic acid as the principal antecedent of uric acid. (Compare pp. 468 and 518.) According to this view, any increase in the number of leucocytes of the blood ought to increase the production of uric acid. As the spleen is generally regarded as in some way connected with the production of the white blood-cells, and as these blood-cells are largely the source of nucleins, it would follow that the spleen is largely concerned in the production of uric acid. Thus, in hypertrophy of the spleen the production of uric acid is increased, and the amount has been found to decrease as the spleen decreases in size. Horbaczewski showed that uric acid is produced when the blood of calves is left in contact with splenic tissue, in presence of oxygen. The amount of uric acid excreted by a healthy man is subject to rather wide variations, ranging from 0.2 to 1 gm. in twenty-four hours. The quantity excreted in the urine is not always an exact measure of the amount produced in the system, for it has been shown that this acid can be, and often is, stored up in the tissues. The formation and the elimination of uric acid are two entirely different things. Both of these factors are of the greatest importance in diagnosis and treatment. Uric acid is a dibasic acid, and hence forms neutral and acid salts. It occurs in the urine as free acid, as neutral sodium and potassium and ammonium urates, and as acid urates of sodium and probably of ammonium. The neutral salts are quite freely soluble in water, while the acid salts are soluble with difficulty, and the free acid is almost insoluble, and appears as a more or less colored sediment. When pure, uric acid forms a white crystalline powder, soluble in 14,000 parts of cold water, and in 1800 parts of boiling water. It is soluble in alkalies, even in the carbonates of the fixed alkalies. The acid is soluble in sodium phosphates, and these salts in the blood serve to hold it in solution in that fluid. It reacts with the sodium phosphate of the blood to produce the neutral or acid urate, according as it is present in smaller or larger quantities:

$$2Na_2HPO_4 + C_5H_4N_4O_3 = 2NaH_2PO_4 + Na_2C_5H_2N_4O_3$$

Or—

$$Na_2HPO_4 + C_5H_4N_4O_3 = NaH_2PO_4 + NaHC_5H_2N_4O_3.$$

The greater the proportion of sodium phosphate in the blood, the greater is the solvent power of this fluid for uric acid. Hence, when we wish to increase the elimination of uric acid, with a given production of it, we do so by increasing the alkalinity of the blood. When the alkalinity of the blood runs down, it is capable of proof that the uric acid is stored in the tissues, to be dissolved out and thrown into the circulation whenever the alkalinity rises again. From these facts it is evident that a single estimation of the amount of uric acid on a given day does not necessarily give the production for that day. Uric acid, when present in excess in the body, gives rise to certain fairly well-described symptoms, such as headaches, muscular pains, neuralgias, nervous irritability, irritable heart with increased blood tension, gout, and nephritic irritation or nephritis. Excess of uric acid excretion may be the result of an increased production, or of a diminished elimination; it is important, from a therapeutic standpoint, for the physician to determine which. This is not always an easy matter. The quantitative estimation of uric acid is time-consuming, and, as we have seen, a number of such determinations are necessary to make these determinations of much value. Persons differ very much as to their tolerance of uric acid, some being very sensitive to a slight increase of it, while others seem to tolerate large quantities without marked symptoms. There is, in health, a fairly constant relation between the amount of uric acid and the urea excreted. The ratio, as stated by different authors, varies somewhat according to experience, and according to the methods used to estimate the urea and uric acid. Some place it as low as 1 to 50 or 60, while others at 1 to 35 or 40. Haig, who examined his urine for 2013 days, found the ratio to be about 1 to 33 or 35. Yvon and Berlioz found it 1 to 30 in one set of experiments, and 1 to 40 in another. Lecanu found 1 to 33. Simon gives it as 1 to 50 or 60, which latter ratio is probably low. My experience with the methods given in this book makes the ratio about 1 to 40. In pathological states it often runs as high as 1 to 20, or in leucocythemia as high as 1 to 12. Uric acid is usually increased in most febrile diseases, as typhoid, pleurisy, pneumonia, pericarditis, rheumatism, etc. It is generally increased in gout, chlorosis, anemia, chronic nephritis, progressive muscular atrophy, and chronic lead poisoning. An increased elimination of uric acid, constituting a disease known as the uric acid diathesis, should be mentioned. Among foods, fresh, butchers' meats produce the greatest increases in the production of uric acid. Such meats contain some uric acid ready formed, but they contain also xanthins, which are the immediate generators of this acid, and hence they introduce uric acid beyond what is produced in the natural course of metabolism. Hence, it is necessary for persons the subjects of the uric acid diathesis to refrain from the free use of such meats. Fats and sugars cause an increased elimination of uric acid. It has recently been shown that a diet rich in nucleins, as thymus, splenic and pancreatic tissue, greatly increase the elimination of uric acid. Tea, coffee, and cocoa are said to increase the production of uric acid, because the caffein which they contain is easily converted into uric acid (Haig).

Of drugs that increase the elimination of uric acid, salicylic acid and its salts, sodium phosphate, salicin, salol, piperazin, quinine, and belladonna deserve to be mentioned.

The drugs that cause a diminished elimination of uric acid are the mineral acids, iron, lead, mercury, silver, copper, zinc, manganese, calcium, opium, cocaine, the iodides, ammonium salts, nitrites, strychnine, and many others. These drugs cause a diminished elimination by forming insoluble compounds with the acid, and holding it back from the blood, while those which increase the elimination do so because of their power of rendering the acid more soluble in the blood, and thus cause this fluid to dissolve it out of the tissues and carry it to the kidneys. It is evident that if these drugs cause an elimination of more than is being formed in the body at the time, their action in this direction can not go on indefinitely, for the supply must soon give out. Such is the clinical result of their long-continued use. For this reason works on clinical medicine usually recommend that in giving the salicylates, if no good results are obtained after two or three days, it is best to try another remedy. Haig states that we may use salicylic acid as a test of the presence of uric acid in the tissues. For, if the administration of a dose of 15 grs. of this acid causes the ratio of uric acid to urea to rise above 1 to 30, we may conclude that there must have been an excess of uric acid in the tissues before its administration. From the above discussion we see that uric acid is a very important constituent of urine, and many disorders have been attributed to its presence in either the blood or tissues. There is a difference of opinion as to whether the acid is more harmful when in the circulation, or in the tissues. Haig claims that it is most injurious when in the blood, while others, among whom is

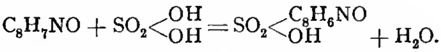
Sir William Roberts, claim that it can produce gouty lesions, at least, only when it precipitates in the tissues.

Pigments.—The normal color of the urine is due to several pigments, chief of which is urobilin. There are two varieties—normal and pathological urobilin. Normal urobilin may be obtained by precipitating it from the urine with basic lead acetate and extracting the color from the precipitate with alcohol; acidifying with H_2SO_4, treating with $CHCl_3$, separating and evaporating off the chloroform. Urobilin is an amorphous, yellow-brown powder. When the urine is treated with ammonia and $ZnCl_2$ solution, it gives a fine green fluorescence. In fever urines it can be precipitated by $NH_4OH + ZnCl_2$. It gives a characteristic spectroscopic absorption band between b and f in the green. Urobilin increases in the urine, on standing, by oxidation of a chromogen or urobilogen. It is derived from both biliary pigments. Some have believed that the urobilin was the same as stercobilin, the coloring matter of the feces. The spectroscopic bands are not the same, however, for the two. It is probable that stercobilin and hydrobilirubin are absorbed from the intestine, and are slightly modified and secreted by the kidneys as urobilin. In febrile diseases the urobilin differs spectroscopically from normal urobilin. The former seems to be a less oxidized stage of the same material than the latter. Baumetz looks upon this substance as an evidence of deficient hepatic acitivity. After extensive extravasations of blood into the tissues of the peritoneum, the urine becomes dark from excess of this pathological urobilin. Urohematoporphyrin is a pigment that has been found by MacMunn and Nobel in the urine of certain diseases, as Addison's disease of the suprarenal capsule, Hodgkin's disease (leucemia), acute rheumatism, cirrhosis of liver, pneumonia, pericarditis, peritonitis, measles, typhoid fever, meningitis. This pigment closely resembles pathological urobilin.

Aromatic substances found in the urine.—The chief of these are indoxyl (sometimes called indican), skatol, phenol, cresol, catechol, and cystin. These aromatic bodies are generated chiefly in the intestinal canal, and are absorbed and eliminated by the kidneys. They exist in the urine, in combination with sulphuric acid and potassium, as ethereal salts. They undergo some oxidation in passing through the blood. Thus, in the case of indol, which is the substance produced in the bowel, it is oxidized into indoxyl:

$$C_8H_7N + O = C_8H_7NO.$$

The indoxyl then reacts with H_2SO_4 as follows:

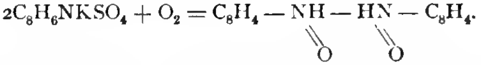


$$C_8H_7NO + SO_2 < \begin{matrix} OH \\ OH \end{matrix} = SO_2 < \begin{matrix} C_8H_6NO \\ OH \end{matrix} + H_2O.$$

This indoxyl-sulphuric acid reacts with N_2HPO_4 as follows:

$$\underset{\text{Indoxyl-sulphuric Acid.}}{C_8H_6NOSO_2OH} + Na_2HPO_4 = \underset{\text{Sodium Indoxyl-sulphate.}}{C_8H_6NOSO_2ONa} + NaH_2PO_4.$$

These aromatic substances are produced as decomposition products whenever proteids undergo putrefactive changes under the influence of bacteria. It has now been pretty well settled that micro-organisms are always concerned in the production of indol, at least, and probably also of skatol and some of the others. The presence of these substances in the urine indicates putrefactive decomposition of proteid matters somewhere in the body, and that the decomposition products have found their way into the circulation. Indoxyl has been studied more than the rest of these aromatic substances, because it is the easiest one to recognize in the urine. Indoxyl is a colorless body, as it occurs in the urine, but it is easily converted into indigo-blue by oxidizing agents. (See p. 638.)



$$2C_8H_6NKSO_4 + O_2 = C_8H_4 - \underset{\substack{\backslash\backslash \\ O}}{NH} - \underset{\substack{\backslash\backslash \\ O}}{HN} - C_8H_4.$$

The amount of indoxyl present may be roughly estimated by the depth of the blue color. For the purpose of making the color more evident we may, and usually do, add a little chloroform, which dissolves the indigo and carries it down with it as it separates. Sometimes a red color (indigo-red, urrhodin, or indirubin) instead of indigo-blue is obtained, and this colors the chloroform red or violet. from a mixture of the two colors. The indigo-red can be dissolved out with ether. Sometimes the urine remains red after shaking with chloroform and ether, due to a red pigment produced by oxidation of skatoxyl. Just what conditions lead to the production of indol, skatol, or phenol have not been determined. According to Filati, indol is produced only in the small intestine, while skatol is produced in the large intestine. This idea, however, is contradicted by the fact that when an artificial anus is established near the lower end of the ileum, the indican and ethereal sulphates almost disappear from the urine, and reappear when the connection between the small and large intestine has been reëstablished. This skatol—red coloring matter—is easily separated from the urine, after removing the indoxyl with chloroform, by pouring off the urine and shaking this with amyl-alcohol, when the pigment dissolves and rises to the top. It is very desirable that we should have further studies of this substance.

The detection of phenol is not often possible without first distilling it off, after making the urine acid with H_2SO_4. This is somewhat tedious, and is not very often done, more especially as we do not know the conditions that develop it.

As we have already said, the presence of indoxyl generally shows that somewhere in the body putrid decomposition of proteids is taking place. The most likely as well as the most frequent place where this occurs is in the intestine. We know, also, that the intestines, in health, always contain micro-organisms. When absorption is retarded or in any way seriously interfered with, or if the digestion of the proteids is incomplete or greatly delayed, these organisms feed on the unabsorbed matters and develop indol, which is absorbed and appears in the urine. Under such circumstances the amount of indoxyl found in the urine is a measure of the amount of putrefaction of the proteids of the food in the intestine. The largest amount of indoxyl secreted, in health, is found after a meal rich in animal food, especially of the fresh meats; it is smallest during a milk diet, or a diet of kephir, kumiss, or buttermilk. Indeed, we can reduce the amount of intestinal fermentation to a minimum by a milk diet. Pot-cheese has also been found to be very effective. Anything that retards peristalsis, as acute and chronic peritonitis, and especially ileus or acute obstruction of the bowel, will increase the indoxyl in the urine. In intestinal obstruction, this may be of great assistance in making a differential diagnosis, as in obstruction of the small intestine the amount of indoxyl is very great, while in disease of the colon it is seldom or never increased.

Allen McLane Hamilton has recently discussed the relation of indoxyl to mental diseases, and concludes that intestinal decomposition has a marked influence in producing certain forms of insanity, especially cases of rapidly developing delusional insanity. He regards the test for indican as of great importance in all insanity cases. He confirms what has been stated by others, that some forms of melancholia are due to auto-intoxication, or the absorption of the products of putrefaction from the intestines ("N. Y. Medical Journal," October 31, 1896).

Simon has noticed that there is a close relationship between the amount of HCl secreted by the stomach and the amount of indoxyl found in the urine. In general, when there is no absorption of decomposed pus anywhere in the body, and there is no stenosis of the small intestine, and the person is taking a normal diet containing no excess of red meats, a decrease of HCl is accompanied by an increased amount of indoxyl in the urine. Exception is made in hysterical hypochlorhydria, and the same condition associated with organic disease of the stomach. In hyperchlorhydria, except in cases associated with ulcer of the stomach, there is usually a diminished excretion of indoxyl. This substance, therefore, has, according to this author, a new significance. Very large quantities are excreted in such diseases as carcinoma of the stomach, and other diseases that produce a diminished secretion of HCl, as acute and chronic gastritis and gastric catarrh.

In all cases in which the peristaltic movement of the intestine is impeded, as in obstinate constipation, obstruction, peritonitis, ileus, etc., indican is increased. It is not necessarily increased in simple constipation without other disease. When it is found in such cases, we should look for disease of the stomach, possibly carcinoma or catarrh. As albuminous putrefaction can take place within the body outside of the intestine, as in empyemia, putrid bronchitis, gangrene of the lung, and in old abscesses, we may expect to find indoxyluria in such cases. Of course, it may be found in pyemia and septicemia. Indoxyl is found in excess in most cases of Bright's disease, but it is probable that it is here only as a result of the digestive disturbance that is associated with this disease. Its presence in epileptic subjects just before an epileptic seizure has frequently been noticed. This fact has led some to believe that there is some relation between the intestinal fermentation and the seizures. From my own experience, I am inclined to think that some attacks may be averted by a brisk mercurial purge, if the intestinal disturbance is recognized in time.

Indican is increased in most cases of pernicious anemia, chorea, and chlorosis. In some cases of melancholia, and even in many cases of insanity, indican is increased, and the symptoms are greatly improved by intestinal antisepsis and cathartics.

The preformed sulphates, as well as the ethereal sulphates, are increased by a diet rich in animal foods, especially the red meats, as well as by intestinal fermentation and proteid decomposition. As the two kinds of sulphates increase and decrease from this cause in about the same ratio, some writers, as Bernacki, Hoppe-Seiler, Baumann, and Rovighi, hold that an increase in ethereal sulphates may mean nothing unless the ratio of these to the preformed sulphates is disturbed. The normal ratio is about 1 of the ethereal to 10 of the preformed sulphates. In cases of marked intestinal fermentation the ratio sinks to 1 to 2, or 1 to 3. Others prefer to be guided by the actual increase of the ethereal sulphates. A very practical limitation to the use of such information lies in the fact that it takes considerable time to make these determinations. This is out of the question with a busy practitioner, even if he possess the requisite skill.

Urohematin is the name sometimes applied to indigo-red. When nitric acid is added to urine containing it, a deep-red or mahogany color is produced. This is sometimes spoken of as **Rosen-**

bach's reaction. It should be regarded in the same light as a highly increased amount of indoxyl. **Uroroseinogen** is another red pigment that has been found in the urine, but its significance has not been made out. **Urohematin, hematoporphyrin,** and **urohematoporphyrin** have also been described as occurring in urine. They are derived from blood-coloring matter. Hematoporphyrin is a color that is found occasionally in the urine of persons who are taking salol. Hematin and hemoglobin are found in urines in malaria, and in some other diseases accompanied by destruction of the red blood-corpuscles. The coloring matters in such cases respond to the guaiacum and turpentine test, but the sediment does not show blood-corpuscles under the microscope.

Toxicity of Urine.—Toxic substances of a basic nature have been found in traces in normal urine. In pathological states, however, and especially in some febrile diseases, such as typhoid, pneumonia, pleurisy, cholera, typhus fever, and acute yellow atrophy of the liver, large amounts may often be found. These substances are mostly diamins, and are to be found among the products of albuminous putrefaction. Bouchard pointed out that these substances are probably produced in the lower part of the intestinal tract. The two diamins that are most commonly found are putrescin and cadaverin. Ptomaines have been frequently found in the urine of maniacs, a confirmation of the notion that auto-intoxication with substances absorbed from the intestines plays an important part in the etiology of insanity. It has long been known that even normal urine is poisonous when it is introduced into the circulation of man or of the lower animals. It has been determined by experiment that the toxicity of the urine of the same individual is not always the same. Bouchard's work on the subject has thrown much light on our knowledge of the toxicity of the urine. He details a series of experiments made by injecting varying amounts of urine into the circulation of healthy rabbits, with careful records of the results.

The symptoms he observed are: 1. Contraction of the pupils of the eyes. 2. Shallow and accelerated breathing. 3. Increase of urinary secretion. 4. Fall of temperature. 5. Diminution of the reflexes. 6. Convulsions, usually followed by coma and death.

He calls that quantity of urinary poisons capable of killing a rabbit weighing 1 kilogram the **urotoxic coefficient.** The normal urotoxic coefficient in man is about 0.465—that is, each kilogram of body weight secretes in each twenty-four hours enough poison to kill 0.465 kilogram of rabbit or of living matter. In other words, each pound of our bodies will furnish enough poison in twenty-four hours to kill 0.465 pound of our body, and hence, in about two and one-sixth days, it will furnish enough to kill itself.

This agrees very well with the time a man usually lives during total suppression of urine. Three days is seldom exceeded, although some elimination by the skin or intestine is usual.

He determined that the cause of the toxic action of urine is principally in the urinary solids. Urine secreted in the waking state is more toxic than that secreted in sleep. The latter is convulsive in its effects, while the former is narcotic. The convulsive agents are principally the mineral salts.

Bouchard has shown that putrefaction in the intestine has a great influence upon the toxicity of the urine. In the case of a man, the subject of gastric trouble, he found that 30 to 40 c.c. of urine induced death of 1 kilogram of animal; while, after disinfection of the intestines with naphthalene, 90 to 100 c.c. proved harmless. On discontinuing the naphthalene, the toxicity of the urine returned. He also found that the administration of charcoal in large doses greatly reduces the toxicity of the urine without especially checking the putrefaction in the intestines. He demonstrated that filtering a toxic urine through charcoal reduced the toxicity of this fluid. Experiments showed that the aqueous extract of putrid proteid matter is very toxic, while that of fecal matter is slightly so. The alcoholic extract of putrid matter is not very toxic, while that of the feces is very toxic. The experiments of Bouchard seem to establish beyond doubt that the cause of the toxicity of the urine lies chiefly in the potassium salts. Potassium salts are forty-four times more toxic than sodium salts. He found that so small a quantity as 0.050 gm. of potassium bicarbonate was enough to kill a kilogram of animal, with violent convulsions, the convulsions coming on after the injection of only 0.030 gm. of the salt. Ammonia is less toxic than potassium, but more so than sodium. One hundred and fifty milligrams of ammonia, combined with carbonic acid, produced convulsions and death of a kilogram of animal. Next to ammonia in importance as a toxic agent come the extractive matters, which are removed by filtration through charcoal. These probably include the alkaloidal substances produced in the intestine by putrid fermentation.

Sugar ($C_6H_{12}O_6$)—*Grape-sugar*, *Glucose*. *Starch-sugar*, or ***Liver-sugar*** (see p. 81).—According to some authorities, glucose is normally present in traces (Brucke and Bence Jones), but according to Seegan, whose investigations are recent and comprehensive, the secretion of sugar is not physiological, and normal urine does not contain sugar. Sugar occurs in large amounts only in the disease known as diabetes mellitus, and hence it is accompanied by the secretion of a large amount of light-colored urine of a specific gravity usually above normal, and having a sweetish odor and taste. Sugar is also found in the urine in certain other affections, especially in disturbances of the abdominal circulation. By wounding certain parts of the medulla, temporary glycosuria may be produced in the lower animals. During the first week of lactation, and after any obstruction to the flow of milk, as after weaning, there is always a small quantity of sugar in the urine, probably lactose. Sugar has appeared in the urine after internal administration of turpentine, nitrobenzene, and nitrotoluene. The liver is continually pouring glucose into the blood to the extent of 1850 grs. per day (4 oz. or 120 gm.), which is burned in the economy. Anything which prevents this oxidation may cause it to appear in the urine. An unusually light-colored or greenish-yellow colored urine, passed in large quantity and of high specific gravity, should always excite suspicion and lead to a test for sugar. A high specific gravity with decreased quantity is frequently met with, which usually contains an excess of mucin, urates, and coloring matter, but no sugar. Such urine will often reduce alkaline copper solutions, but not the alkaline bismuth solution.

Urine containing glucose is somewhat viscid, froths easily, and often has a sweetish odor.

Diabetes Mellitus.—The disease known as diabetes mellitus is characterized by the excretion of an abnormally large amount of urine of high specific gravity and light color, and containing sugar and an excess of urea. The patient usually, if not always, complains of thirst, loss of flesh, and frequent micturition. It is for this last symptom that he usually consults the physician.

The disease seems to be more frequent in England than in the United States. Statistics seem to show that in the former country there are about five deaths in every 100,000 due to diabetes mellitus, while in this country about 2.8 per 100,000. No age is exempt from the disease. It is, however, more frequent between the ages of thirty-five and sixty.

Etiology.—The etiology of diabetes mellitus has been the subject of a great deal of discussion, and many different opinions have been advanced. It is pretty well agreed that the cause of the appearance of sugar in the urine is due to some disturbance of the relation between the sugar-producing and the sugar-destroying functions. C. Barnard has shown that an experimental glycosuria can be produced in lower animals by the irritation of the floor of the fourth ventricle. Cases have been reported in which tumors in this region have led to the production of glycosuria.

An important element in the production of this disease seems to be certain profound disturbances of the nervous system, as great sorrow, nervous shock, etc. This cause, however, accounts for but a part of the cases seen. About fifty per cent. of the cases are associated with disease of the pancreas, as cyst, fatty degeneration, atrophy, calculus, or cancer. This has led to the application of the term pancreatic diabetes to these cases, while those of nervous origin have been termed neurotic diabetes.

A still considerable number of cases do not come under either of the foregoing groups. In many of them the origin and pathology can not be satisfactorily determined. In most cases of diabetes mellitus there are other pathological findings which are not regarded as causes, but as effects. Among these, the liver is frequently found intensely hyperemic. The kidneys become more or less diseased as a result of overwork in the elimination of the large amount of sugar, urea, and water.

Symptoms.—Thirst and frequent micturition are the earlier symptoms noted by the diabetic. In addition to thirst, there is usually an intense dryness of the fauces, tongue, and lips. The saliva becomes viscid and the patient frequently moistens his lips with his tongue. The skin is usually dry and harsh, due to the absence of perspiration. The temperature is normal. Itching of the skin, dyspeptic symptoms, or voracious appetite and constipation are usually observed. Loss of weight and gradual loss of strength are the rule. Less frequent and constant symptoms are cough, tuberculosis, boils and carbuncles, eczema, severe neuritis, most often of the brachial and crural nerves, derangement of the special senses, especially of vision and occasionally of hearing, smell, and taste. The fatal termination of diabetes mellitus is most often preceded by coma, the so-called diabetic coma. Much has been said as to the appearance of acetone and diacetic acid as a forerunner of diabetic coma. More extended researches do not confirm this relation, as acetone and diacetic acid are often present for a long time without the appearance of diabetic coma, and many cases die of coma in which acetone and diacetic acid are not overabundant in the urine.

Tyson says upon this subject that all views as to the cause of diabetic coma are speculative. He believes, with Stadelmann and Minkowski, that diabetic coma is more often associated with hydroxybutyric acid.

Diabetic coma must not be confounded with other forms of coma occasionally met with in diabetes, as, for example, apoplexy and uremia. No doubt many cases which pass for true diabetic coma are cases of uremia, for, as above stated, sooner or later the kidneys are apt to become structurally deranged.

Albuminuria (see p. 74).—Albumin may appear in the urine in a variety of conditions. Albumin is a generic name, and includes a number of more or less closely related bodies. The proteids which may be met with in the urine are serum-albumin, serum-globulin, albumoses (proteoses or propeptones), nucleo-albumin, fibrin, hemaglobin, and histon. Egg-albumin has been claimed by some as an occasional constituent of the urine of persons after eating freely of eggs. Of the different forms of albuminous bodies found in the urine, the most important, from a clinical standpoint, is serum-albumin, and, next to this, globulin and albumose. When the term "albuminuria" is used, it is usually understood to mean serum-albumin in the urine.

Some years ago albuminuria was looked upon as evidence of a serious, diseased condition. In recent years it has been very much discussed whether it may not occur in, and be compatible with, good health. It has been claimed by some that albumin may occur temporarily in the urine of certain persons who are in a state of normal physiological health. This form of albuminuria has been called by them **physiological** or **functional albuminuria.** In some persons the albumin may appear at intervals, and then disappear to reappear again. This form of albuminuria has been called **cyclic albuminuria,** or **intermittent albuminuria.** Some authors regard all forms of albuminuria as abnormal, but they admit that it may appear in cases where no other symptoms of disease can be made out. It is admitted by all that albumin does not appear in the urine as a regular every-day occurrence and without cause. It is certain that very slight causes are sometimes sufficient to determine its appearance; and yet, a normal kidney, without any disturbing element, does not allow albumin or globulin to pass into the urine. The disturbing element is often so slight that many authors are not willing to admit that it constitutes a pathological condition. Such, for example, are: severe muscular exertion, a cold bath, mental exertion, severe emotions, menstruation, during digestion, etc.

Albuminurias are usually discovered accidentally, most often by life-insurance medical examiners. Great care should be exercised in making a diagnosis of physiological albuminuria. For such a diagnosis the amount of albumin must be small, not exceeding one-tenth of the volume of the urine tested; no tube-casts should be present, the amount of urea should be fully up to normal, there should be no retinal changes, no hypertrophy of the left ventricle, no dropsy, and no abnormal pulse-tension.

The albumin is usually absent from the urine passed on rising in the morning, although this is not absolutely necessary. When this does occur, it is a strong point in favor of the diagnosis of functional albuminuria. Such cases should be watched for some time before we decide that we are dealing with a case of "functional albuminuria."

The association of albuminuria with an increased elimination of uric acid and calcium oxalate has been noticed by some authors. There can be no doubt that these substances exert an irritant action upon renal epithelium, and cause disturbance enough to temporarily allow a little albumin to pass through. This albumin can be made to disappear by proper diet and treatment; but, if it be neglected, it will sooner or later cause granular degeneration of the kidneys. While such albuminurias may be only temporary in character, the conditions under which they occur can not be regarded as physiological, although they are not pathological in the sense that there is any evident organic change in the kidney structure.

We may conveniently classify the different forms of albuminuria as follows:

1. *Functional albuminuria,* which has already been described.

2. *Febrile Albuminuria.*—Under this head we would place the appearance of albumin in the course of many of the specific fevers, not dependent upon a true nephritis. The albumin usually appears at or near the acme of the disease, and it disappears during convalescence. In typhoid fever, for example, it is to be expected that albumin will make its appearance in the urine during the height of the fever, and Robin, who has studied this subject, says that it appears in all cases at some time. My own experience does not confirm this statement. I have seen cases in which I have not been able to find it at any time during the course of the fever. As to the cause of the albuminuria, opinions differ. Perhaps it would be better to say that it may be due to several causes, and each of these have been magnified by some one author. The causes that may be mentioned are: Changes in the blood-tension; the irritation of the bacterial poisons; the irritation of the concentrated, highly acid urine, containing an excess of urea, uric acid, and extractive matters; or changes in the composition of the blood itself.

The following notes from Robin's work on "The Urine of Typhoid Fever" will give an idea of the occurrence of albuminuria in other fevers:

Pneumonia: Albumin is usually present as in typhoid. At times abundant, especially in severe cases.

Acute miliary tuberculosis: Albumin not so constant as in typhoid. When present, it is not so abundant as in the severe or fatal cases of typhoid.

Epidemic influenza; grippe: Albumin present in traces in about twenty per cent. of the cases.

Gastric fever: Albumin seldom present.

Herpetic fever; urticaria: Never present in more than traces.

Rubeola: Present only in the severer cases.

Scarlatina: Albumin present in a considerable number of the cases, but usually by virtue of a veritable nephritis.

Enteritis in adults of the adynamic type: Traces usually present.

Cerebro-spinal fever: Albumin present in fairly large amount.

Vegetative endocarditis: Albumin usually present in variable amount.

Acute rheumatism: Albumin present in about forty per cent. of the cases.

Intermittent fever: Albumin present in some cases, but is not constant.

In short, it may be said that albumin may be present in all fevers.

3. *Albuminuria Due to Circulatory Disturbances.*—To this class belong those resulting from cardiac insufficiency due to valvular disease or dilatation, leading to renal hyperemia. The pressure of abdominal tumors or of a gravid uterus, violent exercise, and other causes of disturbed renal circulation may cause albuminuria.

4. *Toxic Albuminuria.*—This has already been referred to as a possible explanation of the albuminurias of the specific fevers. Some authors claim that the albuminuria of pregnancy is due in part, at least, to changes in the blood during this condition. Some of the functional albuminurias are due to changes in the composition of the blood, and Semmola and others believe that in Bright's disease the first change is a blood change. Clinically, we observe albuminuria of toxic or hemic origin in scurvy, purpura, leukemia, pernicious anemia, and in poisoning with cantharides, mustard oil, turpentine, carbolic acid, salicylic acid, petroleum, lead, mercury, copper, iodine, phosphorus, arsenic, antimony, alcohol, the poison of syphilis, uric acid, diabetes mellitus, and after inhalations of ether and chloroform.

5. A *neurotic albuminuria* has been described by some. Found after epileptic seizures, in delirium tremens, neurasthenia, migraine, and Basedow's disease (exophthalmic goitre). This may be a useful test for malingerers. (Senator.)

6. A *digestive albuminuria* must be recognized. This is occasionally seen after the free indulgence in eggs or beef, and it has been seen after drinking freely of root-beer and ginger-ale. English and Frank found albuminuria in two-thirds of the cases of obstruction of the bowel, the quantity being in proportion to the severity of the disease. This disappeared with the relief of the obstruction.

7. *Albuminuria from Organic Disease of the Kidneys.*—In acute nephritis albuminuria is constant, but the amount is subject to considerable variation. The quantity is usually proportional to the

intensity of the disease, from a daily amount of from 5 to 15 or 20 gm.

In chronic parenchymatous nephritis the amount of albumin is usually rather large, and its presence is very constant. In the chronic interstitial form the presence of albumin is not so constant, and the quantity is small. Indeed, cases have been reported in which albumin was never found, and the diagnosis was confirmed by an autopsy. Nor are cases rare in which no diagnosis of nephritis is made before death, it being made only at the autopsy.

In chronic amyloid degeneration the amount of albumin is small, but it is more constant than in chronic interstitial nephritis.

8. *Accidental Albuminuria.*—By this term we will designate albumin added to the urine with pus, blood, leucorrhœal discharge, blennorrhœa, spermatic fluid, lymph, or chyle. Whenever albumin is found in the urine, it becomes an important matter to determine its source and meaning. This is not always an easy matter. We must also remember that we may have a mixed origin of the albumin ; a part of it may come from the kidneys, and a part of it may be accidental. In most cases a careful microscopical examination will reveal its source, for pus, blood, vaginal discharge, spermatic fluid, urethral discharge, and chyle will be made evident at once. In cases of doubt it wi'l be necessary to obtain the urine with a catheter, or even by catheterization of the ureters. We can frequently eliminate leucorrhœal and urethral discharges by instructing the patient to pass a part of the urine before collecting the sample, so as to flush the passage and wash out the mucus and pus.

Serum-globulin frequently occurs together with albumin, in cases where the latter appears, although in smaller quantities. In amyloid degeneration, according to Senator, the proportion of globulin to albumin is much greater than in any other disease. In this disease, he says, the two forms occur in nearly equal quantities, and he thinks such a ratio is fairly constant and of diagnostic importance.

Albumosuria (Peptonuria) (see p. 78).—The occurrence, in urine, of a proteid which is not precipitated by heat and acids has long been known. This substance was formerly known as peptone, or the substance produced by the action of the digestion upon albumin. The reactions by which peptone was shown to be present in urine were, the absence of coagulation with acids and heat, by salt solutions, and by acetic acid and K_4FeCy_6. With our increased knowledge of the peptones and albumoses we have discovered a method of separating these two classes of bodies by means of ammonium sulphate. When this test is applied to urines formerly supposed to contain peptone, we find that, after removing the proteids precipitated by complete saturation with ammonium sulphate, we can rarely find peptone in the solution. What was formerly described, then, as peptonuria is now known as albumosuria, and it is doubtful whether peptonuria really exists. All discussions of peptonuria to be found in the older writings must be regarded as applying to albumosuria. It is probable that albumose occurs more frequently than is supposed, owing to the fact that we do not find it unless we search for it by a special method. It is, therefore, usually overlooked. It has been found to occur in a variety of diseases, as dermatitis, intestinal ulceration, hepatic abscess, croupous pneumonia, septicemia, peritonitis, apoplexy, acute pleurisy, puerperal parametritis, endocarditis, caries, typhoid fever, nephritis, phthisis, measles, scarlatina, leucocythemia, urticaria, acute yellow atrophy of the liver, suppurative meningitis, and in various psychoses. From this array of diseases in which it has been found, it would seem that it can not be highly diagnostic of any. It was formerly stated that the presence of peptone in the urine was associated with suppurations, where there was absorption of the pus or the products of its decomposition. This form was called pyogenic peptonuria. A hepatogenic, an enterogenic, a hematogenic, and, finally, a renal and a vesical form were recognized. The presence of albumose, according to Senator, may be useful in making a differential diagnosis between suppurative meningitis and the simple variety.

Hematuria and Hemoglobinuria (see p. 80).—In this condition the urine usually has a red or brown color, and deposits a red sediment. Hematuria can not be regarded, usually, as a disease, but as a symptom of some other disease. Nevertheless, it is very important that we should be able to determine the source of the blood—whether it comes from the kidneys, tubules, the ureters, pelvis of the kidney, bladder, urethra, or possibly from the rectum or vagina.

We may divide the hematurias, for convenience, into the following classes: (1) **Ordinary hemorrhage from rupture of blood-vessels**; (2) **parenchymatous hemorrhage**, or slow escape of blood through the epithelium of the tubules of the kidney; (3) **hemoglobinuria**, or **hematinuria.**

1. In this variety the urine is dark-red in color, similar to venous blood. Reaction neutral or alkaline; rapidly deposits a sediment, leaving the clear urine above; we often find clots or fibrinous masses; specific gravity variable, but usually high; albumin always present.

2. Reddish-brown, often coffee-colored; retains its uniform color for a long time, but finally deposits a red sediment; generally acid; specific gravity variable. It contains altered hemoglobin or methemoglobin. The sediment is characteristic, when examined microscopically. The blood-corpuscles are not distributed in *rouleaux*, or rolls.

Some of them are changed in size; some spherical and colored brown, or sometimes quite colorless. We often observe corpuscles only one-half or one-quarter of the usual size, and from that down to mere granules, called **microcytes.** They are considered characteristic of parenchymatous hemorrhage from the urinary passages.

3. Hemoglobinuria (Vogel's hematinuria) is also called dissolved blood. The urine is usually quite dark in color, smoky, or even black. The coloring matter is in solution, the urine retains its color, and does not deposit a sediment of corpuscles. Reaction acid; specific gravity lowered. Usually met with in typhoid or typhus fever, malarial infection, and in smelters suffering from inhalation of arsenuretted hydrogen.

Pus.—*Significance.*—Pus in the urine always indicates a suppurative process somewhere along the urinary tract, except in women, when it may get into the urine from the uterus or vagina. The pus may be derived from any part of the urinary passage.

It is sometimes difficult to determine its source, but the following points will aid in making the diagnosis: In blennorrhea of the urethra, a purulent fluid may be pressed out of the urethra between the micturations, or the first few drops of urine passed will be nearly all pus. In this disease, too, the pus is found in shreds or clots in the urine. In purulent cystitis the other symptoms of the disease will usually be present, as frequent micturition, strangury, etc., and perhaps the last few drops of urine will contain a larger quantity of pus than the rest. The urine is apt to be alkaline, and contains a sediment when passed. There is usually pain in the hypogastric region and scalding pain in passing urine. There is not so much pain, however, as in acute cystitis. In suppuration along the course of the ureter there are usually attacks of slight colicky pains in this region. Suppuration in the kidney may be catarrhal or deep-seated (interstitial). In the first, it is accompanied by less disturbance, and is less protracted. It is also accompanied by a considerable loss of epithelial cells from the tubules. In abscess, the flow of pus is generally intermittent, and there is generally a tumor to be felt over the region of one or both kidneys, usually but one. Suppuration confined to the parenchyma of the kidney, is often accompanied by very slight local symptoms. If the pus comes from the pelvis of the kidney, there are lumbar pains and absence of bladder symptoms. The urine is acid. The presence or absence of renal casts will assist in making a diagnosis. There are seen transitional epithelial cells under the microscope. If from the vagina, there will be leucorrhœal symptoms, abundant pavement epithelial cells, with excess of mucus, and acid urine. Absence of bladder symptoms.

Fibrin occasionally occurs in urine. It is met with in cases of hematuria and in chyluria. It has no pathological significance other than this.

Nucleo-albumin has lately been found in the urine of a number of diseased conditions, and, according to some authors, in most normal urines. This latter statement has been disputed by others.

It is claimed that it is always found in icterus, and it is now admitted that it is a normal constituent of bile. It is precipitated by acetic acid and by trichloracetic acid, and is dissolved by nitric acid. Simon denies that a purely renal nucleo-albuminuria exists. The significance of nucleo-albumin does not seem to have been determined.

Another proteid body, known as histon, has been found in urine by Kolis and Burion, Lilienfeld and Kossel. It is precipitated by alcohol and by ammonia, and it gives the biuret reaction. It is supposed to be derived from the leucocytes of the blood, and has been found in leucocythemia. Its pathological significance, other than this, has not been settled.

RENAL DISEASES ACCOMPANIED BY ALBUMINURIA.

Our knowledge of diseases of the kidney dates back to 1827, when Richard Bright, of Guy's Hospital, London, described the disease. These diseases are usually classed under the indefinite title of "Bright's disease." This title is not sufficient. There are at least five varieties, and some say twelve. I shall classify them as follows: Acute and chronic congestion, acute and chronic parenchymatous nephritis, acute and chronic diffuse nephritis, and amyloid kidney.

Acute Congestion, or Hyperemia.—*Definition.*—An abnormal influx of arterial blood, temporary in character.

Causes.—Irritation or paralysis of vasomotor nerves, first stage of acute nephritis; the poisons of various infectious diseases, as scarlatina, small-pox, measles, etc.; exposure to cold and malarial attacks (least common); irritating diuretics, as potassium salts, cantharides, digitalis, etc.

Anatomy.—Kidney normal in size; capsule not adherent nor thickened; kidney darker than normal and filled with bloody serum.

Microscopical Appearances.—Blood-vessels engorged; epithelial cells swollen and a little cloudy; sometimes extravasations of blood-discs into the tubules, and ecchymotic spots in pelvis of kidney.

Symptoms.—Those of the exciting disease. When due to cold or irritating diuretics, there may be a chill or strangury, great pain in loins or hypogastric region, followed by almost total suppression.

Urine has a high specific gravity and high color, and may or may not contain albumin and blood. As recovery begins, the first urine passed is loaded with urates. The symptoms may last a few hours or several days. The urine may contain blood or hyaline and blood-casts, also urates. Prognosis generally favorable. Occasionally fatal, from continued suppression.

Passive or Chronic Congestion (*Cyanotic Induration*).

Causes.—Any mechanical cause which prevents escape of blood from the kidneys by renal veins, as emphysema, pericarditis, hydropneumothorax, mitral insufficiency. The most common causes are organic heart disease, aneurysm of the arch of the aorta, and dilatation of the right heart.

Appearances.—Kidney large and heavy. If recent, capsule not adherent; if chronic, adherent. Color red in both. If chronic, the kidney is hard and nodular, and may be smaller than normal.

Symptoms.—It is difficult to say how many of the symptoms of persons suffering from this disease are due to it, because it is so often associated with other diseases. A congestion of only a few days' duration does not seem to give rise to any marked symptoms. The symptoms are not very constant. Dropsy, when present, is more apt to appear first in the lower extremities, in congestion, while in chronic Bright's disease it appears first under the eyelids. Cough, with mucous or mucopurulent sputum, sometimes with hemoptysis, is met with in some cases. Headache, dyspnœa, delirium, or vomiting may be looked for. The diagnosis will rest upon the evidences of obstructed venous circulation, and upon the urinary changes. In chronic congestion the urine is much decreased in quantity, with a high specific gravity (1025 to 1030), a dark color, and often loaded with urates. Albumin and blood-corpuscles are quite common. Casts are infrequent, but occasionally a few hyaline, finely granular, or blood-casts may be present. Albumin usually present in small amount. If the obstruction increases, the urine becomes less and less, until it amounts almost to suppression. Uremic symptoms will then appear, with headache, convulsions, or coma.

If congestion continues, it ultimately leads to organic changes and permanent nephritis.

Acute Parenchymatous Nephritis.

Definition.—A disease affecting the epithelium of the tubules, by which the cells become swollen and granular, or detached. Kidney enlarged and light-colored. In mild cases the convoluted, in severe cases the convoluted, the straight tubes, and glomeruli are affected. Albumin is present in the urine in all cases.

Etiology.—Exposure to cold is the most important cause. The specific fevers are the next most frequent cause. Pneumonia, typhus, typhoid, yellow fever; acute yellow atrophy of the liver, scarlatina, diphtheria, pyemia, peritonitis, and poisoning by phosphorus and arsenic are common causes. This is essentially the nephritis of acute fevers.

Urinary Symptoms.—Idiopathic Cases, or those due to Exposure. Urine diminished or suppressed. Specific gravity nearly normal. Albumin usually large, often blood. Casts sometimes few, sometimes abundant. Hyaline, blood-, granular, and epithelial casts. In some cases dropsy, anemia, and loss of appetite are the most prominent symptoms; in others, cerebral symptoms, such as delirium, convulsions, stupor, coma, or persisting vomiting, dyspnœa, and great prostration are the most prominent. Little or no dropsy in some cases.

Secondary Cases.—Urine usually diminished in quantity, albumin abundant; sometimes contains blood. Hyaline and granular casts. Dropsy in post-scarlatinal variety, usually not in others. Cerebral symptoms occur in the severe cases. *Duration* in idiopathic cases is short, where dropsies occur. Secondary cases apt to last for some weeks after primary disease disappears.

Chronic Parenchymatous Nephritis.

Definition.—A disease of the tubules, running a chronic course of months or years. The kidney is large, smooth, white, and often weighs sixteen to twenty ounces. Not a very common disease. May follow the acute form, or may be idiopathic or complicate phthisis. The urine is less than normal in amount. Specific gravity variable—1010 to 1030. Albumin present in considerable amount. Hyaline, granular, or fatty casts. No blood. Generally marked dropsy, dyspnœa at night, dyspeptic symptoms, anorexia, and frequently vomiting. Uræmia, stupor, convulsions, coma, etc., only in severe cases.

Prognosis.—Not good, but better than in chronic diffuse nephritis. Many cases completely recover.

Acute Diffuse Nephritis.

Synonyms.—Acute desquamative, acute tubal, croupous, glomerulo-nephritis, acute interstitial nephritis, or *acute Bright's disease.*

Anatomy.—Kidney large; capsule not adherent; surface smooth. Color may be deep-red and congested, or white, mottled with red spots. Epithelium in tubules and glomeruli are swollen and cloudy. Stroma is infiltrated with serum, leucocytes, and blood.

Etiology.—Most cases are the result of exposure to cold or complicate scarlatina.

Symptoms.—After exposure, a person, previously healthy, is attacked with a chill, fever, pain in back, frequent and painful micturition or suppression. Urine bloody or smoky brown. Albumin abundant.

The urinary examination alone will not, as a rule, distinguish between this and the purely parenchymatous variety of the disease. Indeed, the differential diagnosis is seldom possible.

There may be edema of the lungs or of the glottis, or the serous cavities may fill. Appetite *nil;* nausea, vomiting, and often cerebral symptoms supervene. In the worst cases cerebral symptoms develop early, in which case the patient is apt to die in a few days. Where cerebral symptoms do not develop, the disease may continue for weeks or months, or the patient may recover. Skin pale. Albumin and casts persist for a long time, and after all other symptoms are gone. In a few cases the disease becomes chronic.

Chronic Diffuse Nephritis.—*Chronic Bright's, Croupous, Catarrhal, Atrophied,* or *Cirrhotic Kidney.*

Etiology.—The disease prevails in temperate climates, and is more common in middle-aged men than in women, but is seen at all ages. Persons habitually intemperate, or those having constitutional syphilis, or those suffering privation, are most liable to it.

Symptoms are very different in different cases. (1) Persons may have the disease for a long time without knowing it, the symptoms never appearing until some other sickness attacks them, as a heavy cold or some accident. Some are only diagnosed at autopsy. Or (2), the patient may begin to lose flesh and appetite, or suffer with indigestion. Mental or bodily exertion is an effort. He may become pale and anemic, the skin having a peculiar waxy, white appearance. His urine is of low specific gravity and contains a little albumin at night. This may continue for a long time. Or, he may have sleepless nights, headache, irritable temper, drowsiness, neuralgic pains, or cramps in the muscles of the legs. Retinal hemorrhages or retinitis is common, with permanent impairment of vision. He may suddenly become worse. Headache becomes severe; there may be dyspnea at night, or he may suffer with nausea, vomiting, or diarrhea. He may apparently recover under treatment, and get back to his work for a time. Sometimes convulsions suddenly supervene, the patient becomes unconscious between attacks, and dies comatose. Dropsy may or may not appear. *The urine.*—The quantity of urine in the less severe cases is frequently above normal and contains a little albumin and a few casts. In the atrophic variety we are to expect an increase of urine of low specific gravity, with sooner or later uremic symptoms, often early in the disease.

Albumin occurs regularly and in larger quantity than in chronic interstitial nephritis. The specific gravity is usually low, and the total solids and urea are less than normal. The indoxyl reaction is usually very strong.

The casts are both small and large, hyaline, finely granular, and coarsely granular. There are more or less disintegrated epithelial cells. A careful examination of these fragments of cells, and an estimate of their number, will give valuable information as to the amount of organic destruction going on in the kidney, and is a valuable indication in prognosis (see p. 104).

Chronic Interstitial Nephritis (*Sclerosis*, or *Cirrhotic Kidney*).—This disease is a chronic inflammation of the kidneys, with hyperplasia of its connective tissue, with subsequent contraction.

Etiology not clearly known. Syphilis, alcohol, and lead-poisoning have been mentioned. Gout is one certain cause, and in such cases it is probably due to local irritation of uric acid. The kidneys are small, capsules thickened and adherent, and the color red. There is a general arteriosclerosis and hypertrophy of left ventricle of the heart.

The *symptoms* are very vague. In some cases the patient comes into view as a dyspeptic, or as a sufferer from intractable neuralgia, or occipitofrontal headache, or persistent dyspnea with no lung disease, or dimness of vision, or in uremic coma or convulsions.

The *urine* is increased in quantity, and the specific gravity is usually low—1005 to 1010. There is usually but a trace of albumin, and occasionally it is absent. A few hyaline casts will usually be found, but they often require careful search.

Diagnosis.—Large quantity of urine of persistent low specific gravity, with diminished urea, chlorides, and solids, with a small quantity of albumin and a few hyaline casts, persisting for months or years, and accompanied by increased arterial tension and hypertrophy of the left ventricle — these are the symptoms upon which a positive diagnosis may be made.

The urine is usually clear and transparent, and seldom deposits a visible sediment.

Amyloid Kidney (*Waxy Kidney*, or *Lardaceous Kidney*).

Etiology.—Follows prolonged suppuration, especially of the bones or joints. Syphilis, cancer, or ulceration of the intestines are mentioned as causes.

Symptoms.—It is connected with chronic suppurations. Anemia, dropsy of lower extremities, and ascites are usually present, but uremia is seldom present. As the disease progresses there is great weakness, and profuse, uncontrollable diarrhea and vomiting are frequent.

It is differentiated from chronic parenchymatous and chronic interstitial nephritis by its association with chronic suppurative processes, and from the character of the casts.

The Urine.—This is usually increased in quantity, light in color, contains a considerable quantity of albumin, and a few hyaline casts. These casts are usually notched at the edges, are yellowish-gray in color, and sometimes, though not always, assume a mahogany color when treated with a solution of iodine in potassium iodide.

Pyelitis.—This is a disease characterized by suppuration in the pelvis or tubules of the kidney.

Etiology.—The most common cause is a calculus. Exposure to cold, mechanical injury, tuberculosis, infection from the bladder, or rheumatism may be mentioned as causes. One or both kidneys may be involved. The mucous membrane of the pelvis alone may be affected, or it may involve other structures, giving rise to *pyelonephritis.* When cystitis is also present, it is called *pyelocystitis.* When the ureter becomes obstructed so that the pus can not escape, the kidney becomes distended, and it is called *pyonephrosis.*

Symptoms.—The symptoms of pyelitis are not constant, and sometimes there are no distinct symptoms except urinary findings. If caused by a calculus or cystitis, the symptoms are more marked. When the disease becomes well developed, there is a constant, dull, aching pain in the lumbar region, radiating forward into the groin, thigh, testicles, or penis. There is usually frequent desire to urinate. In pyelonephritis there is apt to be emaciation, typhoid-like fever, and symptoms of uremia or pyemia. In pyonephrosis the dull pain, and the tumor to be felt in the region of the kidney affected, will indicate the diagnosis.

The Urine (see p. 81).—The urine is cloudy in the first three of these affections, and rapidly forms a white sediment of pus-cells. The reaction in pyelitis and pyelonephritis is acid; the specific gravity is normal in the first, and normal or less than normal in the second. In pyelocystitis the reaction is alkaline in most cases, and the specific gravity is normal or lower. Albumin and pus are present in all three affections, as is also globulin. In pyonephrosis the pus and albumin may occur in the urine at times, but may at other times be absent. If a calculus be the cause of the pyelitis, more or less blood may be found in the urine. In the earlier stages the peculiar caudate or spindle-shaped epithelial cells from the pelvis will be found, but later in the disease these may be absent.

Acute Cystitis.

Causes.—Traumatism, gonorrhea, infection from a catheter, calculi, retention of urine, pyelitis, very acid urine.

Symptoms.—Usually begins with a chill, fever, loss of appetite, malaise, pain over the pubes, perineum, groin, and thighs; tenderness in the region of the bladder, strangury, and frequently hematuria.

The urine is lessened in quantity, cloudy, neutral or alkaline in reaction, and sometimes becomes putrid in the bladder. It contains numerous epithelial cells from the bladder, and in very acute cases more or less blood and pus.

Chronic Cystitis.—This may follow the acute form, or it may come on gradually. It is usually due to some obstacle to free voiding of the urine, as stricture of the urethra, or prostatic enlargement, vesical calculus, etc. It may result from a prolapsed or anteverted uterus in females, and tumors, either benign or malignant.

Symptoms.—These are similar to those of the acute variety, though milder.

The urine is usually alkaline, viscid, turbid, and ammoniacal or putrid; the quantity is usually normal and the specific gravity is usually below normal. The urine contains an excess of mucus, pus-corpuscles, an abundance of epithelial cells from the bladder, and crystals of ammonium magnesium phosphate. When the cystitis is due to a calculus, blood corpuscles will at times be found, or there may be marked hematuria. When due to enlarged prostate, there will always be a considerable residual urine in the bladder, even after the patient urinates.

Chronic cystitis is apt, sooner or later, to extend upward through the ureters, and produce catarrhal nephritis or pyelonephritis, described above.

Diagnosis.—Cystitis is likely to require differentiation from *prostatitis*. This affection may be either acute or chronic, and the symptoms resemble those of cystitis. There is frequent and painful micturition, the pain being in the perineum and of a throbbing character. There is less control of the desire to urinate than in cystitis. The pain is worse at the close of urination. The urine in prostatitis contains excess of mucus, with mucous casts of the follicles of the prostate.

TABLE OF WEIGHTS AND MEASURES.

ENGLISH WEIGHTS.

TROY WEIGHT OR APOTHECARIES' WEIGHT (U. S. P.).

℔ *Pound.*	℥ *Ounces.*	ʒ *Drachms.*	℈ *Scruples.*	gr. *Grains.*		gm. *Grams.*
1	12	96	288	5760	=	373.2419
	1	8	24	480	=	31.1035
		1	3	60	=	3.8879
			1	20	=	1.2959
				1	=	0.0648

AVOIRDUPOIS WEIGHT.

Pound.	*Ounces.*	*Drachms.*	*Grains.*		*Grams.*
1	16	256	7000.	=	453.5926
	1	16	437.5	=	28.3495
		1	27.343	=	1.7718

APOTHECARIES' OR WINE MEASURE (U. S. P.).

C. *Gallon.*	O. *Pints.*	f℥ *Fl. Ozs.*	fʒ *Fl. Drachms.*	♏ *Minims.*		*c.c.*
1	8	128	1024	61440	=	3785.
	0.1	16	128	7680	=	473.
		1	8	480	=	29.57
			1	60	=	3.70
				1	=	0.06

IMPERIAL MEASURE,

Adopted by the British Pharmacopœia.

Gallon.	*Pints.*	*Fl. Ozs.*	*Fl. Drachms.*	*Minims.*		*c.c.*
1	8	160	1280	76800	=	4543.5
	1	20	160	9600	=	567.9
		1	8	480	=	28.4
			1	60	=	3.55
				1	=	0.06

METRIC MEASURES.

MEASURES OF LENGTH.

1 Millimeter = 0.001 of a meter.
1 Centimeter = 0.010 of a meter.
1 Decimeter = 0.100 of a meter = about 4 inches.
1 **Meter** = **1.000 Meter** = **39.37 inches.**
1 Decameter = 10.000 meters.
1 Hectometer = 100.000 meters.
1 Kilometer = 1000.000 meters = about ⅝ of a mile.
1 Myriameter = 10,000.000 meters = about 6⅓ miles.

MEASURES OF SURFACE.

1 Centaire = 1 square meter = about 1⅕ square yards.
1 Are = 100 square meters.
1 Hectare = 10,000 square meters = about 2½ acres.

MEASURES OF VOLUME.

1 Cubic centimeter = 0.001 of a liter.
1 Liter (cubic decimeter) = 1000 cubic centimeters.
1 Cubic meter = 1000 cubic decimeters.
1 Cubic meter = 1000 liters, or 1 kiloliter.
1 Cubic meter = 1 stere.

MEASURES OF WEIGHT.

1 Milligram = 0.001 of a gram = about 1/65 of a grain.
1 Centigram = 0.010 of a gram.
1 Decigram = 0.100 of a gram.
1 **Gram** = **1.000 Gram** = about 15½ grains.
1 Decagram = 10.000 grams.
1 Hectogram = 100.000 grams.
1 Kilo(gram) = 1000.000 grams = about 2⅕ pounds.

1 Tonneau = 1000.000 kilos = about 1 ton.

ALPHABETICAL TABLE OF EQUIVALENT MEASURES.

1 Are = 100 sq. meters = 119.6 sq. yards.
1 Barrel (wine) = 1.192 hectoliters.
1 Barrel (imperial) = 1.635 hectoliters.
1 Bushel (dry) = 35.243 liters.
1 Centimeter = $\frac{1}{100}$ meter = 0.3937 in.
1 Cubic centimeter = 16.2 minims = 0.06102 cu. in.
1 Cubic centimeter of dist. water at 4° C. weighs 1 gram.
1 Cubic decimeter (1 liter) (1000 c.c.) of dist. water weighs 1 kilogram.
1 Cubic decimeter (imperial measure) = 61.03 cu. in. = 0.8804 qt.
1 Cubic decimeter (American wine measure) = 33.8 fluidounces, or 1.056 qts.
1 Cubic foot = 1628 cu. in. = 28,315.31 c.c.
1 Cubic foot of water at 62° F. (16.6° C.) weighs 62.32 lbs. av.
1 Cubic inch = 266 minims = 16.386 c.c.
1 Cubic inch of water at 62° F. (16.6° C.) weighs 252.46 grs. = 16.372 grams.
1 Cubic meter (1 stere) = 1000 liters = 35.30 cu. ft.
1 Drachm (troy) = 3.888 grams = 60 grains.
1 Fluidrachm = 60 minims = 3.697 c.c.
1 Fluidounce (imperial) = 28.4 c.c. = 1.7329 cu. in.
1 Fluidounce (wine measure) = 29.57 c.c. = 1.8047 cu. in.
1 Fluidounce of water (wine measure) at 62° F. weighs 456 grains.
1 Fluidounce of water (wine measure) at 60° F. weighs 29.57 grams.
1 Fluidounce of water (imperial) at 62° F. weighs 437.5 grains.
1 Foot (12 inches) = 34.48 centimeters.
1 Gallon (imperial) = 277.27 cu. in. = 4.543 liters.
1 Gallon (wine) = 231 cu. in. = 3.785 liters.
1 Gallon of water (imperial) weighs 10 lbs.; wine, gallon, 8.34 lbs.
1 Grain (troy) = 0.0648 gram.
1 Gram (weight of 1 c.c. of water at 4° C., 39.2° F.) = 15.4323 grains.
1 Inch = 2.54 centimeters.
1 Kilogram = 1000 grams = 2.7 lbs. troy = 2.2046 lbs. av.
1 Liter (see cubic decimeter) = 61.027 cu. in.
1 Meter (one forty-millionth of earth's meridian) = 39.3708 in.
1 Minim = 0.0616 c.c. 1 minim of water weighs 0.95 grain.
1 Ounce (troy) = 480 grains = 31.1 grams.
1 Ounce (avoirdupois) = 437.5 grains = 28.35 grams
1 Pint (imperial) = 20 fluidounces = 567.93 c.c.
1 Pint (wine measure) = 16 fluidounces = 473.15 c.c.
1 Pound (troy) = 5760 grains = 373.24 grams.
1 Pound (avoirdupois) = 7000 grains = 453.59 grams.
1 Quart (imperial), 40 fluidounces = 69.97 cu. in. = 1.1358 liters.
1 Quart (wine measure), 32 fluidounces = 58.30 cu. in. = 0.9463 liter.
1 Ton (avoirdupois) = 2000 lbs. = 29,167 ounces troy = 907.20 kilograms.
1 Tonneau = 1,000,000 grams = 1000 kilos = 2204.6 lbs. av.

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